30 1911

VOL. CXLII, No. 1 JULY, 1911 No. 472

JOHNS HOPKINS HOSPITAL 1911 PERIODICAL

THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES

EDITED BY GEORGE MORRIS PIERSOL, M.D.

POSTOPERATIVE AND POSTANESTHETIC NEURASTHENIAS AND PSYCHOSES. By JOHN K. MITCHELL, M.D. . . . 1

A STUDY OF THE NITROGEN METABOLISM IN THREE CASES OF DUODENAL ALIMENTATION. By MAX EINHORN, M.D., and JACOB ROSENBLOOM, M.D., Ph.D. . . . 7

THE DILATATION TEST FOR CHRONIC APPENDICITIS. By W. A. BASTEDO, M.D. . 11

ACQUIRED DIVERTICULA OF THE SIGMOID, WITH A REPORT OF SIX CASES. By ARTHUR D. DUNN, M.D., and PAUL G. WOOLLEY, M.D. . . . 15

PERFORATING ULCER OF THE DUODENUM: DEATH FROM PULMONARY THROMBUS. By GEO. TULLY VAUGHAN, M.D. . . . 34

PURPURA HÆMORRHAGICA, WITH REPORT OF AN ATYPICAL CASE. By A. C. MATTHEWS, M.D., and H. P. CARPENTER, M.D. . . . 36

NEOPLASMS WITHIN THE SKULL: THEIR EARLY DIAGNOSIS AND SOLE TREATMENT. By TOM A. WILLIAMS, M.B., C.M. . . . 42

REFLECTIONS ON SOME CRITICISMS OF THE PSYCHOANALYTIC METHOD OF TREATMENT. By ERNEST JONES, M.D. (Lond.), M.R.C.P. (Lond.) . . . 47

APLASTIC ANEMIA, WITH REPORT OF A CASE. By RALPH C. LARRABEE, M.D. . 57

GRANULOMA PYOGENICUM (BOTRYOMYCOSIS HOMINIS OF THE FRENCH). By RICHARD L. SUTTON, M.D. . . . 69

GENERALIZED ERUPTIONS OF AN UNUSUAL TYPE, CAUSED BY THE ABSORPTION FROM A BELLADONNA PLASTER AND FROM THE OCULAR INSTILLATION OF ATROPINE. By FRANK CROZER KNOWLES, M.D. . . . 73

INVESTIGATIONS CONCERNING THE VALUE OF THE MICROSCOPIC EXAMINATION OF THE BLOOD FOR BACTERIA. By G. A. FRIED, M.D., and A. SOPHIAN, M.D. . . . 88

DIGESTION IN FEVER. By JOHN BENJAMIN NICHOLS, M.D. . . . 93

HEMORRHAGES OCCURRING WITHIN A BRIEF PERIOD IN A GROUP OF CASES OF PULMONARY TUBERCULOSIS. By CHARLES M. MONTGOMERY, M.D. . . . 98

MOUNTAIN SICKNESS. By AUGUST STRAUCH, M.D. . . . 105

PHILADELPHIA AND NEW YORK
LEA & FEBIGER

COPYRIGHT 1911, BY LEA & FEBIGER. ALL RIGHTS RESERVED. ENTERED AT THE POST-OFFICE AT PHILADELPHIA AS SECOND-CLASS MATTER

PUBLISHED MONTHLY. SUBSCRIPTION, FIVE DOLLARS A YEAR, POSTPAID

New (4th) Edition **Just Ready** **Thoroughly Revised**

A MANUAL OF

PRACTICAL HYGIENE

FOR STUDENTS, PHYSICIANS AND MEDICAL OFFICERS

By CHARLES HARRINGTON, M.D.

Late Professor of Hygiene in Harvard University Medical School, Boston

Revised by MARK W. RICHARDSON, M.D.

Secretary, Massachusetts State Board of Health

Octavo, 850 pages, with 124 engravings and 12 plates. Cloth, $4.50 net

---

PUBLISHERS' NOTE

The new edition of Harrington's standard work on Hygiene will maintain it in the forefront of the immense literature on its subject. This pre-eminence it won by reason of its high authority, its exceptional clearness and its adaptation to the needs of all readers: students, physicians, sanitarians, quarantine officials, building and factory inspectors, school boards, military and naval medical officers, architects, and food analysts.

The volume first considers foods of all kinds, including beverages: preservation, contamination, etc. A chapter is devoted to air, one to soil and one to water. Habitations, schools, etc.; disposal of sewage and garbage; military, naval, marine and tropical hygiene are then covered. The relation of insects to human diseases, hygiene of occupation and personal hygiene follow. The very important subjects of infection, susceptibility and immunity are next discussed, and the book closes with a chapter each on vaccination and smallpox, quarantine, and the disposal of the dead.

A part of the work of revision had been accomplished by the late Dr. Harrington himself, and this as well as the remainder of the book have been brought thoroughly up to date by his successor on the Massachusetts State Board of Health, Dr. M. W. Richardson. The features which characterized the earlier editions, and which won the hearty endorsement of all readers, have been continued in this new and thoroughly revised issue, fresh from press.

PHILADELPHIA
706-8-10 SANSOM ST.

LEA & FEBIGER

NEW YORK
2 WEST 45TH ST.

evised

IE

ain-
This
ncas
ati-
rds,

ces:
soil
urid
cal.
nal
urid
on

ate
ock
sa-
res
se-
ed

r.

CONTENTS

ORIGINAL ARTICLES

Postoperative and Postanesthetic Neurasthenias and Psychoses . . . 1
By JOHN K. MITCHELL, M.D., Physician to the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases.

A Study of the Nitrogen Metabolism in Three Cases of Duodenal Alimentation 7
By MAX EINHORN, M.D., and JACOB ROSENBLOOM, M.D., Ph.D., New York.

The Dilatation Test for Chronic Appendicitis 1
By W. A. BASTEDO, M.D., Assistant Attending Physician, St. Luke's Hospital, New York; Associate in Pharmacology and Therapeutics, Columbia University.

Acquired Diverticula of the Sigmoid, with a Report of Six Cases . . . 15
By ARTHUR D. DUNN, M.D., Professor of Medicine, John A. Creighton Medical College, Omaha, Nebraska, and PAUL G. WOOLLEY, M.D., Professor of Pathology, Ohio-Miami College of Medicine, University of Cincinnati; Director of the Laboratories of the Cincinnati Hospital.

Perforating Ulcer of the Duodenum: Death from Pulmonary Thrombus . 34
By GEO. TULLY VAUGHAN, M.D., Professor of Surgery, Georgetown University; Chief Surgeon to the Georgetown University Hospital; Surgeon to the Emergency Hospital; Consulting Surgeon to the Government Hospital for the Insane, Washington, D. C.

Purpura Hæmorrhagica, with Report of an Atypical Case 36
By A. C. MATTHEWS, M.D., and H. P. CARPENTER, M.D., Assistant Physicians, Hudson River State Hospital, Poughkeepsie, N. Y.

Neoplasms within the Skull: Their Early Diagnosis and Sole Treatment . 42
By TOM A. WILLIAMS, M.B., C.M., Corresponding Member Soc. de Psychol. et Soc. de Neurol. de Paris; Member Associate Soc. Med. Ment. Clin., etc.; Neurologist to Epiphany Free Dispensary, Washington, D. C.

Reflections on Some Criticisms of the Psychoanalytic Method of Treatment 47
By ERNEST JONES, M.D. (Lond.), M.R.C.P. (Lond.), Associate in Psychiatry, University of Toronto; Director of the Ontario Clinic for Nervous and Mental Diseases.

Aplastic Anemia, with Report of a Case 57
By RALPH C. LARRABEE, M.D., Second Assistant Visiting Physician to the Boston City Hospital, Boston, Massachusetts.

Granuloma Pyogenicum (Botryomycosis Hominis of the French) . . . 69
By RICHARD L. SUTTON, M.D., Associate Professor of Dermatology, and Chief of Clinic, University Medical College; Dermatologist to the German Hospital and to the Swedish Hospital, Kansas City, Missouri.

Generalized Eruptions of an Unusual Type, Caused by the Absorption from a Belladonna Plaster and from the Ocular Instillation of Atropine. Review of the Local and General Eruptions Caused by Belladonna and Atropine 73

By Frank Crozer Knowles, M.D., Dermatologist to the Presbyterian Hospital; Assistant in Dermatology, University of Pennsylvania; Assistant Dermatologist to the Philadelphia General Hospital; Dermatologist to the Church Home for Children, to the Baptist Orphanage, to the Dispensary of the Children's Hospital; Assistant Dermatologist to the Dispensaries of the Pennsylvania Hospital and the Howard Hospital, Philadelphia.

Investigations Concerning the Value of the Microscopic Examination of the Blood for Bacteria 88

By G. A. Fried, M.D., Adjunct Consulting Physician, Bedford Sanatarium, Bedford Hills, N. Y., and A. Sophian, M.D., George Blumenthal, Jr., Fellow in Pathology, Mt. Sinai Hospital, New York.

Digestion in Fever 93

By John Benjamin Nichols, M.D., Lecturer on Dietetics, George Washington University; Pathologist, Episcopal Hospital; Attending Physician, Freedmen's Hospital, Washington, D. C.

Hemorrhages Occurring within a Brief Period in a Group of Cases of Pulmonary Tuberculosis 98

By Charles M. Montgomery, M.D., Instructor in Medicine, University of Pennsylvania; Physician to the Home for Consumptives, Chestnut Hill, Pa.

Mountain Sickness 105

By August Strauch, M.D., Chicago, Illinois.

REVIEWS

Modern Treatment; The Management of Disease with Medicinal and Non-medicinal Remedies. By American and English Authorities. Edited by Hobart Amory Hare, M.D. 119

Dislocations and Joint Fractures. By Frederic J. Cotton, A.M., M.D. . 121

Operative Surgery. By John J. McGrath, M.D. 124

Bismuth Paste in Chronic Suppurations. By Emil G. Beck, M.D. . . 124

Die Wassermannsche Reaktion mit Besondere Berücksichtigung ihrer Klinischen Verwertbarkeit. Von Privat-Dozent Dr. Harold Boas. . 125

Difficult Labor. A Guide to its Management. By G. Ernest Herman, M.B., F.R.C.P. 125

Practical Obstetrics. By E. Hastings Tweedy, F.R.C.P.I., and G. T. Wrench, M.D. 126

Medical Electricity and Röntgen Rays, with Chapters on Phototherapy and Radium. By Sinclair Tousey, A.M., M.D. 127

PROGRESS OF MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF

WILLIAM OSLER, M.D., W. S. THAYER, M.D., AND R. S. MORRIS, M.D.

A Sulphur Reaction in the Urine in Cancer . . . 129
Diagnostic Value of the Butyric Acid Test (Noguchi) in Cerebrospinal Fluid . . . 130
Peptid-splitting Ferment in the Saliva . . . 131
Cardiac Insufficiency, Albuminuria, and Cylindruria Caused by Chronic Coprostasis . . . 131
A New View of the Function of the Renal Glomerulus . . . 132
The Sensibility of the Alimentary Canal in Health and Disease . . . 132
The Relationship of Electrocardiographic Methods to Medicine . . . 134
The Behavior of Various Organic Acids Concerned in Diabetic Acidosis . 134
Urobilinogenemia . . . 134

SURGERY

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D., AND T. TURNER THOMAS, M.D.

Jejunal Ulcer after Gastro-enterostomy . . . 135
The Anatomical Basis for Surgery of the Lymph Nodes; The Regeneration and New Formation of the Same . . . 135
Suction Treatment in Sterile Sand Baths of Infected Wounds and Wounds Suspected of Being Infected . . . 136
The Operative Treatment of Varicocele . . . 137
The Prognosis of Dislocations of the Shoulder and of Posterior Dislocations of the Elbow . . . 138
A Contribution to the Question of Coxalgia, Coxa Vara, and Juvenile Osteoarthritis Deformans . . . 138
Injury as a Causative Factor in Cancer . . . 139
Gastro-enterostomy as Shown by the X-rays . . . 139

THERAPEUTICS

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.

The Treatment of Acidosis . . . 140
Silver Nitrate Irrigations in Treatment of Infectious Diarrhea in Children 140
The Vaccine Treatment of Typhoid Fever . . . 141
An Analysis of the Clinical and Serological Results Obtained in the Treatment of Syphilis with Salvarsan . . . 141
The Use of Digipuratum in Heart Disease . . . 142
A Safe and Rapid Method for Antityphoid Vaccine . . . 143
The Injection of Drugs, Especially of Salvarsan, into the Lumbar Muscles 143
The External Application of Magnesium Sulphate in the Treatment of Erysipelas . . . 143
The Treatment of Pernicious Anemia with Salvarsan . . . 144
Adalin, a New Hypnotic . . . 144
Serum Treatment of Hemophilia . . . 144

PEDIATRICS

UNDER THE CHARGE OF

LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.

Total Congenital Absence of the Teeth 145
Chronic Lung Affections in Children 145
Ephemeral, Traumatic Glycosuria in the Newborn 146
Petechial Hemorrhages from Artificial Stasis as a Diagnostic Help in Scarlet Fever 146
Confirmation of Rumpel and Leede's Phenomenon in Scarlet Fever . . 147
Edema during Gastro-intestinal Conditions in Infants 147

OBSTETRICS

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.

Our Recent Advances in Obstetric Surgery of Proved Value 148
Spontaneous Gangrene in both Ischiorectal Fossæ 149
The Repair of Vesical Vaginal Fistula after Hebostiotomy 149
Myoma Complicating Pregnancy 149
Advanced Extra-uterine Pregnancy 150
Momburg's Method for Controlling the Circulation in the Pelvis . . 150

GYNECOLOGY

UNDER THE CHARGE OF

JOHN G. CLARK, M.D.

Sacro-iliac Relaxation 151
Treatment of Cystocele 151
Relation of the Thyroid to the Ovaries 152

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES

UNDER THE CHARGE OF

J. SOLIS-COHEN, M.D.

Vincent's Angina Involving the Larynx Exclusively 153
Diverticulum of the Trachea 153
Intralaryngeal Actinomycosis 154
Laryngostomy in Perichondritis of the Larynx 154
Paralysis of the Recurrent Laryngeal Nerve 154
New Oral Speculum for Direct Examination of the Mouths of the Eustachian Tubes and Parts Adjacent 154
Auto-inspection of the Vocal Apparatus during Phonation 154
Grave Hemorrhage after Tonsillectomy 154

PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF

WARFIELD T. LONGCOPE, M.D.

Experimental Contribution to the Study of Cardiac Hypertrophy . . 155
The Passage of Antitoxic Sera into the Cerebrospinal Fluid 155
The Value of the Treatment of Acute Articular Rheumatism with the Antipyretics 156

CaB245433

THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES

JULY, 1911

ORIGINAL ARTICLES

POSTOPERATIVE AND POSTANESTHETIC NEURASTHENIAS AND PSYCHOSES.

By John K. Mitchell, M.D.,

PHYSICIAN TO THE PHILADELPHIA ORTHOPEDIC HOSPITAL AND INFIRMARY FOR NERVOUS DISEASES.

There is not and there cannot well be any reliable evidence as to the frequency with which neurasthenias or other forms of mental disturbance follow operations, since the operating surgeon often knows nothing of their occurrence, many of them not making their appearance until weeks or even months after surgical and anatomical recovery is complete. The surgeon's records, therefore, only show such acute troubles as passing deliria, temporary confusion, or aberration, such as sometimes appear directly after operations, and may with probability be referred rather to the anesthetic or to the physical shock than to the operative procedure; some examples of this type are recorded in the accompanying list.

For the purposes of the present discussion, I have made examination of some hundreds of case histories, taken with few exceptions during eight or ten years past. The investigation was limited to this period for several reasons: (1) Because during this time special attention had been given in my case-taking to those instances in which operation was alleged as among the causes of the nervous trouble; (2) because many of the cases within this time are near enough for memory to confirm or criticise the formal notes; and (3) because the histories are much more minute and complete than those of earlier years.

To make clear the basis of the diagnoses, a brief explanation of terms must be made. "Neurasthenia" covers cases of the fatigue-neurosis type, without marked mental symptoms. "Melancholia" is used to indicate instances of nervous depression, often hypochondriac, not including the more serious forms such as might be put under the head of manic-depressive insanity or the recurrent periodic melancholias. "Psychasthenia" has been applied only to cases with obsessions, or *idées fixes*, and those which though superficially seeming neurasthenic exhibited on further study mental disturbances. These are all "revised diagnoses," recorded when patients were discharged, the original titles having been hysteria, hysteroneurasthenia, hypochondriasis, neurasthenia, cerebrasthenia, myelasthenia, or whatever other diagnostic fragments one might choose to chop the general name of neurasthenia into, and intended in practice to serve as temporary signposts pointing to the therapeusis. So far as possible, instances in which distinct neurasthenia was present before operation have been excluded, so that the form in which the record stands fairly represents careful final diagnostic consideration of various mental, psychical, or neurotic disorders consequent upon, if not causally connected with, surgical procedures. Further, only notes of private patients have been used, not even including those treated in private rooms in hospital service, the former being for the most part under closer observation by myself and by experienced assistants, and their histories not depending in any way upon records made by internes, who, however alert and well-trained, have not the same opportunities of personal study of the patients.

Certain definite forms of neurasthenia and psychasthenia have been omitted from consideration altogether, as those of sexual origin, a few in which it seemed possible that beginning paresis or dementia præcox was at the root of the symptoms, several in which the history was too vague and those in which the patient for any reason was not long enough under observation for the certain establishing of the diagnosis.

344 case histories remained after these exclusions, ranged under the several headings named. These were reviewed, with the aim of discovering how many of them could fairly and reasonably be classed as postoperative or postanesthetic troubles.

Total cases examined 344: Women, 220 (or 64 per cent.); men, 124 (or 36 per cent.).

Instances of neurasthenic or mental disorders following operation or anesthesia, 31 (or 9 per cent. of the total cases examined): women, 29 (or 94 per cent. of the postoperative cases); men, 2 (or 6 per cent. of the postoperative cases).

No.	Social condition.	Age.	Year operated.	Operation.	Notes.
1	Female, married	30	1887	Cervix dilatation	Professor Goodell; panic terror preceding operation; violent hysteria following, lasting many years.
2	Female, married	34	1889	Ovariotomy, double	Professor Goodell; mania immediately following operation; death from exhaustion.
3	Female, single	38	1893	Ovariotomy, double	Neurasthenia following; many years' duration.
4	Female, married	36	1899	Ovariotomy	Nervousness preceded; ovariotomy; stump-abscess complication.
5	Female, single	62	1900	Carbuncle and hemorrhoids	Melancholia followed.
6	Female, married	43	1900	Kidney fixation. Curettage, two operations (one year apart)	Hysterical hypochondriasis.
7	Female, single	28	1901	Ovariotomy	Hysteria.
8	Female, married	31	1903	Cervix repair	Neurasthenia, hypochondriac type.
9	Female, married	36	1903	Total hysterectomy	Mother had puerperal insanity; psychasthenia; obsession of guilt.
10	Female, married	55	1898	Hysterectomy Amputation of breast, two operations (five years apart)	Six months after operation, nervous depression began, lasting five years, then breast amputation, followed by increased depression.
11	Female, single	28	1903	Appendectomy	Bad family history of insanity; acute depression.
12	Female, married	32	1896	Perineum repair, 1896; ovariotomy, 1897; hysterectomy, 1902; three operations years apart	Previous health perfect; neurasthenia.
13	Male, married	..	1904	Appendectomy	Acute insanity of a few weeks' duration.
14	Female, married	..	1905	Total hysterectomy	Psychasthenia followed.
15	Female, married	36	1899	Two operations; one ovariotomy; then hysterectomy and ovariotomy	Doubtful history of nervousness preceding; hysteria following.
16	Female, married	44	1905	Cervix repairs	No ether given, at patient's desire; hypochondriac neurasthenia following directly.
17	Female, married	30	1906	Sebaceous cyst; cervix, kidney fixation	Always nervous, much increased after operation, and finally severe psychasthenia.
18	Female, married	46	1906	Curettage; also two induced abortions	The abortions added a moral cause, operations perhaps only contributed to the sequent neurasthenia.
19	Female, married	38	1899	Curettage; ovariotomy; suspension; three operations	Neurasthenia; previous health perfect.
20	Male, married	33	1907	Appendectomy, 1903; epulid, 1907	Psychasthenia followed first operation, increased by second.
21	Female, married	26	1908	Ovariotomy and nasal septum straightened	Neurasthenia; previous health good; gastroptosis.
22	Female, married	57	1908	Hysterectomy for tumor	Neurasthenia.

No.	Social condition.	Age.	Year operated.	Operation.	Notes.
23	Female, single	50	....	Ovariotomy and several ocular tenotomies	Hysteroneurasthenia.
24	Female, married	40?	....	Ocular tenotomy and two curettings	Increasing neurasthenia.
25	Female, married	..	1909	Cervix repair; curettage	Sudden neurasthenia, with depression six weeks later.
26	Female, single	44	1902	Hysteroövariotomy	Previous health perfect; hypochondriac neurasthenia of very obstinate type; endless doctors, psychotherapy, etc.
27	Female, married	28	1909	Appendectomy; hemorrhoids	Neurasthenia; operation during typhoid attack.
28	Female, married	38	1910	Appendectomy	Psychasthenia, hypochondriac ideas.
29	Female, single	40	1910	Thyroidectomy and two operations on hemorrhoids	Always nervous, but never neurasthenic before operation.
30	Female, married	20	1910	Appendectomy; acute, first attack	Neurasthenia.
31	Female, married	26	....	Ether, no operation	Delusion of criminal assault.

The dates refer to the year in which operation was done, but with few exceptions the histories of the cases were taken during the years from 1898 to 1910.

Of these 31 cases, 29 are from the full case histories, selected by the process of elimination described as fairly representing instances of postoperative trouble. With these 29 are included one case of my own, of which I find no complete statement in my archives, but of which memory supplies a very distinct record, and one supplied by Dr. Weir Mitchell, Cases 2 and 31 respectively.

The operations in these cases varied very widely, from the most trifling surgical intervention, such as ocular tenotomy, straightening of the nasal septum or dilatation of the cervix uteri, up to total hysterectomy and ovariotomy.

The sex distribution is rather striking. Although aware that the proportion of male patients with serious neurotic symptoms was a large one, it was rather unexpected to find it more than one-third of the total. It is interesting to observe that this close equality in the incidence of neuroses is not maintained in the cases suffering from postoperative disorder, in which males have a very small share. The comment of course suggests itself that diseases of the female reproductive organs account for a large number of the operative cases. The males represent but 2 in the total of the 31 instances of postoperative mental disturbances, and these 2 amount to less than 0.5 per cent. of all the cases examined. While not directly connected with the subject, it is worth adding that in the total case histories examined in making up this list the proportion of male cases gave 41 per cent. of the neurasthenias, 25 per cent. of

the melancholias, and 41 per cent. of the psychasthenias, although all instances of sexual neurasthenias and obsessions were strictly excluded.

The worse and most persistent examples of nervous disorder have not by any means always followed the most serious operations. No doubt, as in other neurasthenic conditions, a certain constitutional or temperamental predisposition is required as a precedent, and one cannot make a rule excluding this as one of the causes, as one would in judging of purely traumatic neuroses; but I have tried to exclude from the list those in which there were marked and distinct causes other than operative in addition to the neurasthenic predisposition. Bodily shock or severe pain are not evidently among the necessary causes of this condition. The mental or nervous factors, particularly fright, play a larger part than is generally attributed to them, I feel sure. We practitioners are so accustomed to operations that we do not always allow for the alarm of patients, and treat their fears too lightly.

Complications in the surgical course of the cases seem to have played no part, as in only one instance (No. 4) was any such trouble reported, and in many it was particularly stated that progress had been uninterrupted. Nor can reckless or careless surgery be charged with the unfortunate results, for if it were possible to publish the operators, many distinguished names would be found in the list, whose positions would sufficiently guarantee that care had been used and every precaution taken, both in the selection and treatment of the cases.

Two or three of the cases collected are sufficiently interesting for brief separate comments, as they teach highly instructive lessons.

In one, No. 31, a young woman, a widow, went to a dentist's office with her mother and her attending physician, with the intention of having two teeth extracted under ether. When the administration was begun she struggled and fought so wildly that her mother would not consent that the attempt at anesthization should be continued. The administration ceased before complete relaxation was produced, and no operation of any kind was performed. When the young woman came out of her ether intoxication she asserted with every evidence of the fullest belief that she had been assaulted during her stupor, and this delusion, in spite of every assurance of her mother and of the physician, who had never left her side, persisted very strongly for several weeks, and then gradually faded.

No. 1, seen many years ago with the late Professor Goodell, was etherized for dilatation of the cervix. She was an intelligent woman of education, and had the matter fully explained to her, but was in great fear of the anesthetic. The operation was done briefly, with no violence. When she recovered from the anesthetic

she was in a condition of extraordinarily severe hysteria, and continued for twenty years to exhibit one after another an amazing series of hysterical phenomena; and, indeed, up to the time of her death never recovered complete mental balance and control.

In another case, No. 2, operated upon by Professor Goodell, a difficult ovariotomy with adhesions, requiring rather long etherization, the patient, who had been in a perfectly good mental state before the operation, was wildly maniacal after the ether, and continued in a state of furious mania until her death from exhaustion, about two weeks later.

No special investigation was made in the earlier instances as to the actual time spent in bed after operation, but in the later ones this question has been systematically inquired into, and has usually been answered by the statement that the patient was up at the earliest possible time. I am not unaware that there are good surgical reasons for getting patients out of bed, particularly after abdominal operations, as soon as may be; but I am strongly of opinion that troubles of the kind we are discussing would be less frequent if, instead of trying to get the patient up as soon as possible, the period of rest in bed and mental repose—of course, with general treatment, massage, and perhaps bed gymnastics—were rather to be extended than curtailed, an opinion borne out by personal experience gained in watching the numerous instances in which it has been necessary to have operations performed upon patients either already neurasthenic or at least with the temperamental tendency toward nervous disorders. Under these circumstances, it has been my custom to insist upon a more prolonged rest and isolation than would be considered necessary for purely surgical reasons, and with excellent results in avoiding postoperative nervous complications.

The statements of patients as to physicians' opinions we all know must be received with the utmost skepticism, so that we may to some extent discount the assertions quite frequently made that relief from neurasthenic symptoms had been held out as a reason for operating; but in many instances there has been additional testimony confirmatory of the patient's reports, in some coming from surgeons themselves on this point.

This leads to a warning which may be addressed both to the physician who, desperate over the lack of success of his treatment of a neurasthenic woman, turns for aid to his surgical colleague and also to that invaluable coadjutor. Should the question of surgical treatment of a neurasthenic person arise, much more than the ordinary care must be exercised in deciding upon operation. Wise practice will choose any other route than a surgical one, however roundabout it may make the journey. The only sufficient reason for an operation in a neurasthenic is that the procedure is one which would be absolutely demanded were the patient in

perfect nervous health. The physician and the consulting surgeon must never be deceived into undertaking an operation for the relief of neurasthenic symptoms, unless these symptoms would require, imperatively require, surgical measures in a person not nervous. An operation may relieve neurasthenic symptoms; it will not cure the patient, and there is a good chance that a few months will see her worse than ever, having gained nothing but a new experience, and very likely an exchange of old symptoms for new ones. We should think twice before advising an operation on a neurasthenic, and having thought twice it will often prove best to think a third time, and then not operate. Above all, the operation should never be recommended with the idea or even the hope that if successful it will cure the neurasthenic. The neurasthenia may be no worse after than before; but most assuredly it will very, very seldom be cured.

A STUDY OF THE NITROGEN METABOLISM IN THREE CASES OF DUODENAL ALIMENTATION.

By Max Einhorn, M.D.,
and
Jacob Rosenbloom, M.D., Ph.D.,
New York.

(From the Wards and Chemical Laboratory of the German Hospital, New York City, and the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons, New York City.)

As is well known, it is impossible to maintain the nitrogenous equilibrium in patients kept under rectal alimentation. This deficiency greatly impairs the utility of that method of feeding. One of us has accordingly devised a new mode of feeding subjects in whom absolute rest for the stomach is demanded, viz., by introducing the food directly into the duodenum with a duodenal pump.

By the new method a duodenal pump is inserted directly into the digestive tract and left there for two weeks. As soon as the end of the pump has reached the duodenum, nourishment can be injected into the latter.[1] The food can thus be conveyed into the duodenum at desire and the stomach can be kept entirely free from contact with the food.

While clinically this method of feeding was found to be wholly

[1] A full description of the method of introducing the food, and of the clinical history of the patients whose nitrogen metabolism was determined under this method of feeding, may be found in the following papers: Einhorn, Zeit. f. Physik. und diät. Therapie, 1910, xiv, 452; Interstate Med. Jour., 1910, xvii, 758.

TABLE I.—Miss S. Typical case of gastric ulcer. The weight of the patient at start of treatment was 116½ pounds. The duodenal pump was inserted on April 14, 1910, and feeding was started on April 15. The pump was removed on April 28. The weight at the end of the treatment was 114 pounds.

Date, 1910.	Total nitrogen.			Urine.								Feces.	
						Urea N.		Ammonia N.		Uric acid N.			
	Ingested.	Excreted.	Balance.	Volume.	Total N.		Percentage of total N in urine.		Percentage of total N in urine.		Percentage of total N in urine.	N	Percentage of total N ingested.
	grams.	grams.	grams.	c.c.	grams.	grams.		grams.		grams.		grams.	
April 20	16.2	11.47	+4.73	1370	9.97	8.3	83.25	0.445	4.47	0.35	3.51	1.5	9.26
April 21	15.9	13.11	+2.79	2115	11.61	9.71	83.64	0.474	4.09	0.42	3.61	1.5	9.43
April 22	15.8	13.47	+2.33	1620	11.97	10.04	83.88	0.290	2.42	0.37	3.09	1.5	9.49
April 23	15.2	14.92	+0.28	1480	13.42	11.98	81.82	0.962	7.18	0.48	3.58	1.5	9.87
April 24	16.0	16.48	—0.48	1120	14.68	12.54	85.42	0.502	3.42	0.36	2.45	1.8	11.25
April 25	15.2	13.34	+1.86	1030	11.54	9.96	86.31	0.368	3.13	0.30	2.60	1.8	11.84
April 27	16.3	16.34	—0.04	1250	14.54	12.60	86.66	0.52	3.57	0.37	2.54	1.8	11.04
April 28	16.4	19.21	—2.81	1340	17.41	14.52	83.45	0.62	3.56	0.41	2.35	1.8	10.98
Totals	127.0	118.34	+8.66										
Average	15.875	14.792	+1.08										

TABLE II.—Mr. B. Typical case of gastric ulcer. The weight of the patient was 132 pounds on June 25, 1910. The tube was inserted on June 23, and feeding was started on June 24. The tube was removed on July 7. At the end of the treatment the patient weighed 132 pounds.

Date, 1910.	Total nitrogen.			Urine.						Feces.	
						Urea N.		Ammonia.			
	Ingested.	Excreted.	Balance.	Volume.	Total N.		Per cent. of total N. in urine.		Per cent. of total N. in urine.	N.	Per cent. of total N. ingested.
	grams.	grams.	grams.	c.c.	grams.	grams.		grams.		gms.	
June 28	17.2	16.58	+0.62	1450	15.08	12.55	83.2	0.78	5.2	1.5	8.72
June 29	16.2	16.46	—0.26	980	14.96	12.64	84.5	0.73	4.9	1.5	9.25
June 30	15.9	15.22	+0.68	1420	13.72	11.89	86.7	0.70	5.1	1.5	9.43
July 1	17.4	16.36	+1.04	1640	14.86	12.75	85.8	0.70	4.7	1.5	8.62
July 2	16.4	16.33	+0.07	1280	14.83	12.79	86.1	0.78	5.3	1.5	9.14
July 3	16.4	15.27	+1.13	890	14.07	11.85	84.2	0.65	4.6	1.2	7.31
July 4	16.2	16.40	—0.2	1380	15.20	12.74	83.8	0.62	4.1	1.2	7.41
July 5	14.5	16.12	—1.6	1900	14.92	12.64	84.7	0.60	4.7	1.2	8.27
July 6	16.4	15.66	+0.74	1810	14.46	12.30	85.1	0.71	4.8	1.2	7.31
July 7	16.1	16.08	+0.02	1280	14.88	12.63	84.9	0.76	5.1	1.2	7.45
Totals	162.7	160.48	+2.22								
Average	16.27	16.048	+0.222								

TABLE III.—Mr. F. Typical case of gastric ulcer. The weight of the patient at the beginning of the treatment was 146 pounds; at end, 146 pounds. The tube was inserted on June 14, and withdrawn on June 28.

Date, 1910.	Total nitrogen.			Urine.						Feces.	
						Urea N.		Ammonia.			
	Ingested.	Excreted.	Balance.	Volume.	Total N.		Per cent. of total N. in urine.		Per cent. of total N. in urine.	N.	Per cent. of total N. ingesetd.
	grams.	grams.	grams.	c.c.	grams.	grams.		grams.		gms.	
June 17	15.5	16.33	—0.83	595	14.93	12.80	85.8	0.48	3.2	1.4	9.03
June 19	15.5	15.16	+0.34	565	13.76	11.30	82.1	0.51	3.7	1.4	9.03
June 20	16.4	16.80	—0.4	630	15.40	13.16	85.4	0.48	3.1	1.4	8.54
June 21	16.3	13.51	+2.79	445	12.11	10.40	85.9	0.47	3.9	1.4	8.59
June 24	16.4	15.31	+1.09	650	13.91	11.60	83.4	0.62	4.4	1.4	8.54
June 25	16.6	14.16	+2.44	730	12.86	11.08	86.2	0.38	3.0	1.3	7.83
June 26	16.4	14.70	+1.7	870	13.40	11.40	85.1	0.58	4.3	1.3	7.93
June 27	16.2	14.22	+1.98	860	12.92	10.98	84.9	0.54	4.2	1.3	8.02
June 28	16.0	12.72	+3.28	940	11.42	9.71	85.0	0.56	4.9	1.3	8.12
Totals	145.3	132.91	+12.39								
Average	16.144	14.767	+1.376								

satisfactory, it remained to be seen whether a perfect nitrogenous equilibrium could be maintained, and whether the protein food was assimilated in a normal manner. In order to decide these points we have made analytical studies of the nitrogen intake and output in three patients who have been kept on duodenal feeding, each for a period of two weeks.

All of the patients suffered from ulcer of the stomach. In one the ulcer was situated near the cardia. In the two others the ulcer was situated in the lesser curvature of the stomach, and was complicated with pylorospasm, leading to temporary ischochymia. As a rule, we gave eight ounces of milk, one raw egg, and one or two tablespoonfuls of lactose, the mixture being well mixed and strained before introduction. At each feeding the patient received the above amount of food, at intervals of two hours, eight times daily. When there is intolerance to this quantity of nourishment for each feeding half the quantity may be given every hour. This was the case in one of our patients throughout the treatment, and in another at times. In one of them (Mr. F.) the lactose had to be omitted on account of the great irritability of the enteric tract on account of an existing colitis.

Tables I, II and III contain the results obtained by the examination of the urine and feces of these patients. Samples of the milk given to these patients were analyzed for its daily nitrogen content. The nitrogen of the weighed eggs (minus shell) was computed with the aid of the figures given by Koenig (2.008 per cent. N). The feces were marked off by means of carmine into periods of four to five days.

Nitrogen determinations were made by the Kjeldahl method, the ammonia by the Folin method, urea by Benedict's new method (*Jour. Biolog. Chem.*, 1910, viii, 405) and uric acid by the Folin-Schaffer method.

TABLE IV.—Summary of data pertaining to total nitrogen metabolism (Tables I to III).

	Ingested grams.	Excreted grams.	Balance grams.
Table I	127.0	118.34	+8.66
Table II	162.7	160.48	+2.22
Table III	145.3	132.91	+12.39
Average	145.0	137.24	+7.76
Average daily	16.11	15.25	+0.86

It can readily be seen that in these three cases of duodenal alimentation the absorption of the nitrogen of the food took place to a normal extent. Nitrogenous katabolism, as measured by the quantities of urea and ammonia that were excreted, was normal in degree. All of the patients had a positive nitrogen balance throughout the duodenal feeding periods.

THE DILATATION TEST FOR CHRONIC APPENDICITIS.

By W. A. Bastedo, M.D.,

ASSISTANT ATTENDING PHYSICIAN, ST. LUKE'S HOSPITAL, NEW YORK; ASSOCIATE IN PHARMACOLOGY AND THERAPEUTICS, COLUMBIA UNIVERSITY.

That many persistent digestive disturbances are manifestations of a latent or chronic appendicitis has been repeatedly demonstrated by the disappearance of the disturbances after the removal of the appendix. It is also well-known to operators that in some of these cases the appendicitis was not recognized for a long time, and that even after long observation there were cases in which there was a large element of uncertainty as to whether the appendix was involved or not. In other words, the appendicitis was latent, and could not be detected by the ordinary means of examination. Hence any measure by which such a latent appendix involvement can be recognized deserves consideration. We would, therefore, again call attention to the usefulness of dilating the colon with air to determine the presence or absence of a latent or chronic appendicitis. Since our first report of the test, we have applied it in a large number of abdominal cases, and have been able in a number of instances to establish the diagnosis of appendicitis when all other methods of examination failed completely or left the examiner in a state of reasonable doubt. In addition, we have received verbal reports from several surgeons who have been employing the test as a routine in their hospital cases. In their experience, as well as our own, the test as checked by operation has proved reliable, failure being reported in only four or five cases in several hundred.

To make the test a colon tube is passed eleven or twelve inches into the rectum and air injected by means of an atomizer bulb. If, as the colon distends, pain and tenderness to finger-point pressure become apparent at McBurney's point, there is appendicitis. We have compared the test in a number of instances with the Rovsing test and find this much the more certain; but at times, after moderate dilatation with air, the Rovsing method of forcing the air back into the cecum may be used with advantage. We might sound a warning that if most of the air is not allowed to escape before withdrawal of the tube, colicky pains are likely to ensue.

The test is not needed in an acute case, and in such would be contraindicated; neither is it required in an undoubted chronic case. But the indication for the test is a suspected chronic or latent appendicitis, or any persistent digestive or abdominal disturbance, in which no cause can be found for the trouble. Ordinarily one may entertain doubt about the diagnosis, or at least hesitate about urging operation, when tenderness at McBurney's point can be

elicited only on very deep pressure, or is accompanied by a similar tenderness elsewhere in the abdomen. At times, for example, we have thought of appendicitis because of McBurney's point tenderness, but have found in addition puzzling points of tenderness along the transverse colon or at a spot on the left side corresponding with McBurney's. In such cases, dilatation frequently results in the disappearance of all the points of tenderness except that at McBurney's, which it intensifies.

Again, in persistent cases of hyperchlorhydria or gastrosuccorrhea the test should be performed. For just as in the case of a gall-bladder, so a latent appendicitis may have its chief manifestation in stomach derangement, even so marked at times as to simulate an ulcer. And since it has become our routine practice to dilate the colon in all long-standing cases of the kind, we have had the good fortune in a number of instances to discover an unsuspected appendix and to see the gastric symptoms disappear with the removal of the offending vestige.

A further application of the test may be to distinguish between an inflamed appendix and a right-sided pelvic trouble. Pain and tenderness in a right-sided chronic salpingitis or cystic ovary sometimes result from the colon dilatation, but the tenderness is regularly less acute, is low down in the abdomen, and extends toward the middle line. In three instances we have been able to diagnosticate pelvic inflammation in young women in whom appendicitis was suspected and in whom a vaginal examination was impossible except under ether. In each of these the subsequent operation revealed a cystic right ovary and a free uninvolved appendix. We have employed the test in not a few other gynecological cases, and while in some we have been able merely to corroborate the findings of a vaginal examination, in others we have demonstrated appendicitis in addition to the pelvic lesion. In every such case operated upon the finding of the dilatation test has been found correct.

A few typical case reports may be of interest:

CASE I.—G. L., painter, has had attacks of pain in the abdomen at intervals for one and one-half years, without nausea or vomiting. Recently such attacks have become more numerous, and in the last, he had to lie down for one afternoon; he was told he had painter's colic. He told us that the pain occurred mostly just above the umbilicus or high up beneath the right ribs. He had no lead line on the gums, no polychromatophilia in the blood. On colon dilatation, the gall-bladder was not made out, and no pain appeared in the hepatic region; but in two spots there were pain and tenderness, one spot just at McBurney's point and another just below the navel. A small umbilical hernia also made its appearance. The patient was advised to have an operation for the hernia and the appendicitis, but as the diagnosis was not confirmed by others he

was treated for three months for lead poisoning, intestinal indigestion, and rheumatism. The attacks, however, increased and were more localized in the appendix region, so he returned for operation. The dilatation test was again positive, and operation was performed by Dr. H. H. M. Lyle. The chronically inflamed appendix was covered by veil-like adhesions, which extended to the hernial opening; the gall-bladder contained no stones. The appendix and the adhesions were removed and the hernia closed. The patient has had no more attacks of the old type, and a little recurrence of the pain beneath the right ribs disappeared quickly under treatment for hyperchlorhydria.

CASE II.—D., a physician, in two years had five attacks of severe pain in the abdomen, with prostration and vomiting. The pain was always diffuse, never localized, and lasted about one day; the temperature never rose above 99° F., and the pulse was normal or slow. Physicians had suggested appendicitis, but no positive diagnosis was made. Two days after the last attack, which was so severe that morphine had been administered, the patient walked to my office apparently well. Slight tenderness to finger-point pressure at McBurney's point could be elicited only on very deep pressure, but on dilating the colon an acute pain appeared in the appendix region, and tenderness over an area as large as a silver dollar and centring over McBurney's point. Four days later, Dr. J. A. Blake operated and found a chronically inflamed appendix with a constriction close to the cecum, and adhesions extending upward over the cecum. The patient has had no attack since the operation (not quite a year).

CASE III.—Mrs. R., twelve years ago, had an attack of pain in the abdomen, with vomiting, and was in bed one day. A surgeon saw her in the attack, and said it was not appendicitis. During the entire twelve years since then she has taken a laxative pill every night and has had no further severe pain, but for the last six months has been losing appetite and becoming more costive, and has been irritable and in low spirits. A month ago had a little abdominal pain on the right side for one day, but not enough to require treatment. A test breakfast showed free hydrochloric, 48; total acidity, 70. No organic acid. On dilatation of the colon, pain at McBurney's point with sharply localized tenderness became manifest. It was our belief that hyperchlorhydria treatment would be futile in the presence of a chronic appendicitis, so operation was advised, and Dr. L. W. Hotchkiss removed a retrocecal swollen appendix with three marked constrictions and surrounded by adhesions. Since the operation, eight months ago, the patient has had unusually good digestive and bowel functions and has been in excellent general health and spirits.

CASE IV.—Miss H., aged twenty-four years, a rather underdeveloped young woman, with a mitral stenosis, has had in the

last year several attacks of cramp-like pain in the right iliac region. Two or three times this pain came at the menstrual period, but occurred also at other times. Vaginal examination was not feasible, so the colon was dilated. At once there was a dull pain over the whole lower right segment of the abdomen, extending from McBurney's point to Poupart's ligament and to the midline. Tenderness was slight, and was most pronounced about half way between McBurney's point and the symphysis pubis. Operation by Dr. H. T. Goodwin showed a right ovarian cyst and a normal appendix.

CASE V.—Miss G., aged twenty-seven years, has had pain in the right side low down for a year or more. It has never been very acute, never caused vomiting, and was most pronounced after the patient had been a long time on her feet. There has been a rather abundant vaginal discharge. Examination per vaginam reveals a tender boggy mass in the right fornix, and much tenderness when the uterus is moved. Out of curiosity, the colon was dilated, and to our surprise an acute pain appeared in the appendix region, and tenderness localized at McBurney's point. Operation by Dr. Frank Markoe showed right salpingitis with tube, ovary, and chronically inflamed appendix bound together in a mass of adhesions.

CASE VI.—D. S., has never had any acute attack of appendicitis, but has had some pain in the appendix region when his bowels seemed full of gas. Dilatation was positive for appendicitis. Some time later, in Chicago, he had an acute attack which was diagnosticated appendicitis, and though prostrated and with fever, he took train immediately for New York. Dr. H. H. M. Lyle operated and found a retrocecal abscess with a sloughed off appendix.

These cases illustrate the positive findings of the dilatation test. In the use of the test during the last four years we have had no case in which the test was positive and the operation findings negative. But in two out of all of our cases, the negative finding of the test was followed within six months by a typical attack of appendicitis, and the test was, therefore, presumably at fault. Several times in the early days of the test surgeons operated for a suspected appendicitis, though the test was negative, and in every such case the appendix was found normal. We have had a verbal report from one surgeon, who has used the test extensively, of two cases which gave positive test but negative findings at operation. With very few exceptions, therefore, the test has proved accurate, and it may well serve as a guide to the necessity for operation, particularly in the three classes of cases mentioned, viz., suspected chronic appendicitis, persistent gastro-intestinal or abdominal disturbance with unknown cause, and appendicitis versus ovarian or tubal inflammation.

ACQUIRED DIVERTICULA OF THE SIGMOID, WITH A REPORT OF SIX CASES.[1]

By Arthur D. Dunn, M.D.,

PROFESSOR OF MEDICINE, JOHN A. CREIGHTON MEDICAL COLLEGE, OMAHA, NEBRASKA.

AND

Paul G. Woolley, M.D.,

PROFESSOR OF PATHOLOGY, OHIO-MIAMI COLLEGE OF MEDICINE, UNIVERSITY OF CINCINNATI; DIRECTOR OF THE LABORATORIES OF THE CINCINNATI HOSPITAL.

At the Twenty-seventh Congress of Surgery, Graser demonstrated a stenosis of the sigmoid due to chronic hyperplastic inflammation, on which an operation for cancer had been performed. He believed the portal of entrance for the infection to be a number of acquired diverticula, some of which were seen entering the inflammatory mass. From Graser's paper dates the clinical interest in the subject, for before that time diverticula had been considered merely pathological curiosities. They had been described by Morgagni (1796), Fleischmann (1815), Albers (1844), Habershon (1857), Klebs (1869), Loomis (1872), Fischer (1900), and others.

Acquired diverticula are of two varieties, the true and the false. True diverticula are merely wide-mouthed pouches whose walls contain all of the layers of the normal bowel. They are the result of an abnormal tendency to haustrum formation, and seem to be of anatomical significance only. False diverticula, on the contrary, are herniaform protrusions of the mucous membrane through the muscularis, and are of increasing clinical and pathological moment.

Acquired diverticula may occur throughout the entire intestinal tract. They have frequently been seen in the duodenum in the vicinity of Vater's papilla. They are common in the colon, and they cease abruptly at the first portion of the rectum. Occasionally they have been observed in the appendix, where they may be the seat of a condition which cannot be differentiated from appendicitis. They vary in number from several hundred (400, Hansemann) to an isolated protrusion. They are generally multiple.

Since diverticula of the sigmoid are much more likely to give rise to clinical manifestations, your attention will be directed toward them only.

Etiology. Their origin is still a mooted question. A predisposition to diverticulum formation undoubtedly exists, and according to Graser and Sudsuki is common. On microscopic examination of the sigmoid in old subjects, the former found small diverticula in 10 out of 28 cases, and the latter in 15 out of 40 cases.

[1] Read before the Cincinnati Society for Medical Research, February 24, 1910.

The following theories as to cause have been suggested:

1. That the fenestra through which the vessels penetrate the muscularis form *loci minoris resistentiæ*. The vessels are surrounded by loose connective tissue which contains a variable amount of fat. As the individual ages, the fat increases, the connective tissue becomes weaker, and the fenestra are increased in size by atrophy of the muscularis. Intermittent stasis also stretches and increases the diameter of the fenestra. During periods of cessation of stasis, the caliber of the vessels diminish, and loose space in the "vessel holes" results. These factors increase the predisposition. Accumulation of feces and gas incident to constipation, bowel atony, and abnormal decomposition act to a degree as determinining factors. The vascular theory is accepted by Fischer, Koch, Hansemann, Berner, and others, and is corroborated by four of the cases which we studied microscopically.

2. Bland-Sutton believes that the appendices epiploicæ form points of lessened resistance because, as he has shown, their fat is in immediate continuity with the subserous fat of the intestinal wall. It is difficult to see, however, why continuity of fat should predispose to diverticula. The process is that of a penetration of the muscularis, and it seems highly improbable that the weak serosa, and its subserous layer of fat, could play any part in it. It is more probable that the fact that bloodvessels penetrate the muscularis at this point is the chief reason for the common incidence of diverticula here. In some cases an adherent appendix may cause the production of traction diverticula.

3. Franke believes that developmental anomalies are at the basis of diverticulum formation. What these developmental imperfections are he leaves unspecified.

4. Beer and Telling consider weakening of the muscular coat in adult life as the chief cause of diverticula. They do not state why the muscular wall should weaken in certain spots and not in others. They assume that the vessels may "direct the path" for the diverticula. Beer states: "The fact that diverticula occur in old people, in people whose intestines have been more or less worked out, as evidenced by constipation, points to a muscular deficiency, and in this muscular weakness the cause of false diverticula must be sought."

5. Age seems to be to some extent a determining factor. In 80 cases the average age was sixty years (Telling). Forty-seven of the 80 cases gave rise to clinical symptoms at the average of fifty-five years. The ages of our 6 cases were as follows: Without symptoms, 75, 78, and unknown; with symptoms, 83 (symptoms due to cicatricial stenosis secondary to diverticulitis), 45, and 50.

6. Sex also plays a part. Of 81 cases, 53 were male, 28 female (Telling). Our 6 cases were all men.

7. Obesity has been emphasized as a causative factor by Klebs, Mayo, and others, but it seems to be only an indication of involution

processes and thus only a concomitant of weakness in the muscularis or in the "vessel holes." Only 2 of our cases showed any unusual increase in fat.

8. Cachexia and absence of fat were marked in one of our cases. It might be inferred that in individuals who had been obese the loss of fat would tend to weaken the intestinal wall by leaving unoccupied or loose spaces.

9. The physiological role of the sigmoid must be of some moment in promoting diverticulum formation. The feces are retained longest in this segment (average ten to twenty-four hours), and its walls are incessantly subjected to changes in pressure due to variations in the quantity and quality of its contents, and so a predisposition and tendency to atony is inherent in the normal function of the sigmoid.

10. When the physiological retention of feces becomes excessive and constipation arises, the strain upon the gut walls is abnormal and any tendency to atony is aggravated. Localized weakenings are subjected to a determinative factor. The causal relationship of constipation to diverticulum formation so often observed might seem to support this view. Fischer's doubts as to the importance of constipation, however, are well founded. Its role has certainly been exaggerated and cases are numerous in which constipation has not been recorded. Only a very small proportion of the constipated have diverticula. Abnormal decomposition of retained contents with gas production is probably of more moment. Constipation, if it plays a part, must apparently be of the atonic type. The writers know of no investigation as to this point. Any factor, however, that will increase intrasigmoidal pressure must influence the development of diverticula at points of lessened resistance.

We may briefly summarize the etiological factors, all of which may be present to a variable extent in every case, as follows: (1) Congenital or acquired anatomical predisposition to localized weakness in the wall of the gut. (2) The addition of certain positive mechanical factors to the above potential factors, viz., (*a*) Strain on the bowel wall due to the accumulation of gas and feces. (*b*) Changes in the caliber and structural strength of the "vessel holes" consequent on stasis and senile circulatory disturbances, or on rapid loss of visceral fat. (*c*) Atony, which may be due to age, distention, or obesity.

Pathology. There is little definite information on the postmortem incidence of acquired diverticula of the sigmoid, for, as a rule, little attention is given to this segment of the gut in the routine autopsy, unless clinical manifestations or conspicuous pathological alteration draws the attention thither. In 78 cases in which the sigmoid was carefully examined by us at the autopsy, acquired diverticula were found five times. Two of the above cases were clinical. In one, the diagnosis was suspected almost to a certainty,

in the second, it was a "find." The remaining cases were discovered because they were looked for.[2] False diverticula are usually situated near the mesenteric attachment. According to Bland-Sutton, Franke, and Telling, they are most commonly formed at the appendices epiploicæ. In three of our autopsies they were located at these points. In one case their location was at the mesenteric attachment. In one case, the diverticula were located in the vicinity of the appendices, but their relationship to them could not be determined on account of the hyperplasia of the gut wall and an inflammatory infiltration of the immense amount of fat surrounding the sigmoid. One peculiarity in this case not hitherto noted is the location of the diverticula on the summits of the rugæ, where they were discovered as minute depressions only after careful search. One of these innocent-looking depressions led into a fistulous tract. Location opposite the mesenteric attachment has been described by Hansemann.

The *size* of *acquired diverticula* varies from minute herniaform protrusions of the mucosa, discoverable only with the microscope, to processes the size of peas or grapes. (Size of an apple—Edel; of an egg—Virchow). They are conical, sacular, spherical, or teat-like processes

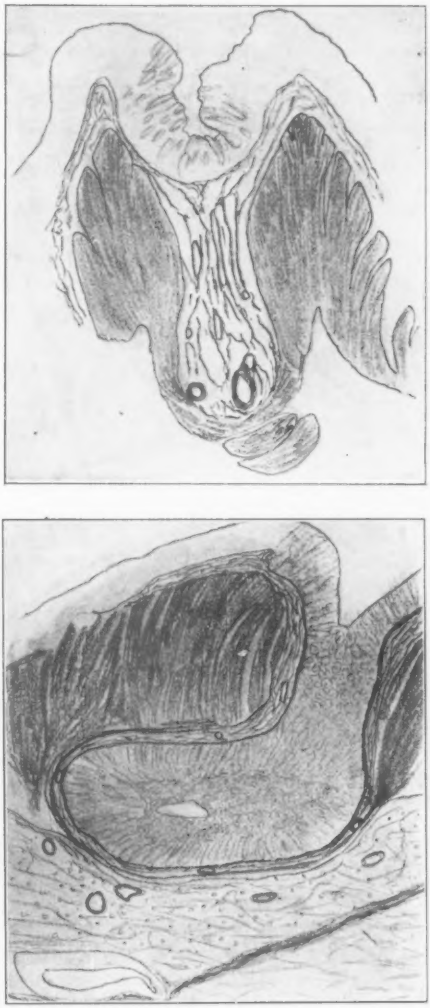
The ostia vary markedly in size. Sometimes the diameter of the opening equals that of the average diameter of the diverticulum. Often the openings are narrow, and although one easily discovers the pouch on the outer surface of the bowel, the mucosa must be scanned carefully to find the mouth. Many of them are distinctly flask-shaped, and their necks seem to be constricted by the penetrated musculature. This shape is especially characteristic of diverticula into the appendices epiploicæ, and creates an excellent mechanism to prevent drainage. Catarrhal inflammation may easily cause edema and swelling of the mucosa, and thus completely occlude the neck. Thus, there is formed a thin-walled sack filled with infectious material lying outside of the gut. The peritoneal cavity is protected only by a thin membrane consisting of mucosa, serosa, and possibly a few attenuated muscle fibers. The mucosa itself is often eroded or atrophic. It may be completely denuded. Localized foci of infection are common.

The coats of the diverticulum vary. Mucosa, submucosa, and serosa are constant. The amount of muscularis varies in different diverticula, and in different parts of the same diverticulum. The walls are thinnest at the tips of the processes.

Here there may be nothing but a thinned outer layer of mucosa and serosa. In our sections the penetration of the muscularis is clear, even though the diverticulum carries some muscle fiber with

[2] Since the above was written one case has been found in the pathological service of Dr. Woolley in the Cincinnati Hospital, the first observation in 103 autopsies. No symptoms were referable to the diverticulum.

it. The contrast in the thickness of the muscular coating of the diverticulum, and of the layers through which it passes, is most



FIGS. 1 and 2.—Sketches (camera-lucida) to show the development of false diverticula.

marked (Figs. 1, 2, and 3). This accumulated musculature at the neck is suggestive of a sphincter. Beer and others have suggested

that the origin of this picture is a localized weakening of the muscularis, but this still leaves an explanation to be explained. The potentiality for mischief which lies in these structures is evident from an inspection of the sections shown with the description of our cases.

The morbidity from false diverticula depends on the following factors:

1. Thinning of the coats.
2. Ulcerative and perforative action of retained concretions and feces.
3. Presence of pathogenic microörganisms.
4. Defective drainage due to: (*a*) Lack of an effective muscular coat. (*b*) Closure of the neck by edema of the mucosa secondary to colitis or stasis. A mild catarrhal colitis may occlude the openings, and thus result in the formation of a pocket of infectious

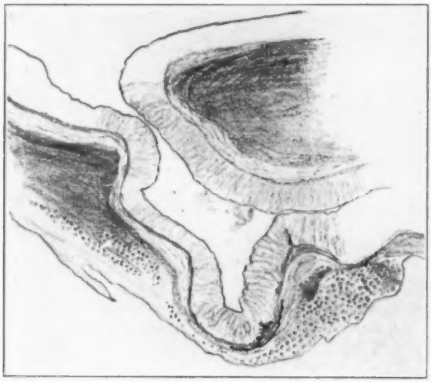


FIG. 3.—Sketch (camera-lucida) to show the development of a false diverticulum.

material, from which the peritoneal cavity is poorly protected by a coat of variable thinness. (*c*) Strangulation of the opening by kinking, torsion, or muscular (sphincter) action.

The following morbid processes, due to acquired diverticula of the sigmoid have been noted:

1. *Local Peritonitis.* Cases have been reported in considerable number. Abscesses in the left iliac fossa have frequently been drained and attributed to various causes, such as left-sided appendicitis, infected appendices epiploicæ, colonic ulceration, etc. This is probably the most frequently observed clinical manifestation of diverticulitis. (Telling collected 24 cases.) One of our cases was of this type.

2. *Acute General (Diffuse) Peritonitis.* (*a*) With perforation, 14 cases (Telling). (*b*) Without perforation. Loomis (1872) reports

a single case of general peritonitis in which diverticula were present in large numbers. He assumed that the infection traversed the thin wall of a diverticulum without rupture.

3. *Chronic Hyperplastic Sigmoiditis.* Within the last few years a number of cases have been reported, characterized by a productive inflammation of the sigmoid, which manifests itself by disturbed function of the lower bowel; pain, intermittent and colicky in character; tenderness; and constipation, often spastic in type and frequently alternating with diarrhea. Mucus and occasionally a trace of blood are present in the stools. Examination showed tenderness in the left iliac fossa with a movable sausage-shaped tumor. Recent observations prove a large percentage of these cases to be the result of diverticulitis and peridiverticulitis. Patel says: "Peute-être un jour sera-t-on droit de dire que dans presque tous ces cas decrites sous le nom de sigmoiditis, il s'agit de véritables divérticulites, absolument comme l'appendicite a été substituée peu à peu à la typhlite." There is little question but that many of the successful resections of the sigmoid for carcinoma have been cases of infiltrating sigmoiditis. A review of museum specimens (Mayo, Wilson, Telling, etc.), is already putting certain excised cancers of the sigmoid into this class. Such reports will increase in frequency as the condition becomes better known.

4. *Cancer.* Four cases are reported by Hochenegg, Stierlin, Telling, and Wilson,[3] in which cancer has developed on the basis of a sigmoiditis and perisigmoiditis secondary to chronic diverticulitis. The low grade of prolonged inflammatory irritation which so often precedes carcinoma elsewhere is here produced by a chronic diverticulitis.

5. *Stenoses.* These are the result of inflammation in the intestinal wall. Usually they are caused by a productive inflammation, *i. e.*, sigmoiditis and perisigmoiditis which narrows the lumen of the gut. The mucosa is thrown into folds, but, as a rule, is not ulcerated. Cicatricial contraction following a diverticulitis may produce stenosis, as is clearly shown by one of our specimens. Telling reports the only other case in the literature.

6. *Adhesions.* Adhesions produce symptoms by: (*a*) Traction and constriction. The obstruction is commonly due to the fact that the small bowel becomes adherent to the inflammatory area. (*b*) Adhesions of the gut to the bladder with vesicosigmoidal fistula. Telling found adhesions reported sixteen times and fistula eleven times.

7. *Chronic Mesenteritis.* Riedel, Brehms, Ries, Simpson, and Gardinier have called attention to the probable importance of sigmoid diverticulitis in the production of retraction and shortening

[3] Donald MacCrae, of Council Bluffs, in a personal communication, reports another case in which he excised a carcinoma of the sigmoid which was penetrated by numerous diverticula and which showed the carcinoma developing in inflammatory tissue.

of the sigmoid mesentery, a state which predisposes to volvulus. One of our cases was of this type.

8. *Metastatic Suppuration.* Whyte (1906) has reported the only case of this type. A limited necropsy disclosed multiple abscesses of the liver. The original focus was supposed to be an inflamed diverticulum of the sigmoid containing a fecal concretion.

9. *Perforation Into a Hernial Sac.* Stierlin has reported a case of suppuration into a hernial sac from a perforated diverticulum in an incarcerated sigmoid.

CASE I.—B. G., male, aged forty-five years. The family history is negative, and the personal history also until one and one-half years before the present illness, when the patient had an attack of pain and distress in the hypogastrium, with temperature. There was some diarrhea and tenesmus. Constipation was not present at any time. He went to bed for several days on a restricted diet and recovered rapidly.

Except a few fleeting pains and an occasional sense of fulness in the lower quadrants, which disappeared with catharsis, nothing of moment occurred for one and one-half years, when, after a few days of indefinite distress in the hypograstrium, he suffered severe pains in the lower abdomen, which were accompanied by dizziness, faintness, chill, temperature of 103°, and later sweating. The bowels had been free, but with some griping and soreness. The patient went to bed on a restricted diet, and for several days had an irregular temperature, ranging from 99° to 103.5°, accompanied by distress in the lower abdomen. Examination brought out the following facts:

The patient was a well-developed middle-aged man. His complexion was muddy, his tongue coated, and his breath foul. Examinations of the heart, nervous system, and lungs were negative except for the rapidity of the heart and for signs of retraction at the left apex. The abdomen was uniformly distended and tympanitic. There was diffuse tenderness below the umbilicus, which was best defined in the left iliac fossa, and in the median line, where he always referred his pain. No tumor mass could be demonstrated. A slight visceromotor reflex was present in both lower quadrants, but was most marked on the left. There was no area of cutaneous hyperesthesia. Neither testicle was tender. Rectal examination was negative. The leukocyte count was 15,000. The urine contained much indican, but was otherwise negative. The stools were very offensive, contained a little mucus, but neither blood nor pus. Large thick Gram-positive bacilli were present in large numbers. The pulse was rapid (100 to 130).

With colonic irrigation and on a diet of buttermilk and carbohydrates the patient slowly improved. For a month there were occasional attacks of pain, with a little temperature, which were relieved by flushings. Convalescence was slow. Several leukocyte

counts during this period gave an average of 8000 to 10,000. Indican was more or less constantly present until convalescence was well established. A diagnosis of intestinal toxemia with possible sigmoiditis was made at that time.

The patient left home and did fairly well for one month, when he was taken with sudden, severe pain in the hypogastrium, hurried to the hospital, and was operated upon with the diagnosis of appendicitis. In a personal communication, the surgeon stated that "the appendix was thickened and slightly injected, but I must confess it hardly seemed sufficiently involved to account for the large amount of turbid fluid which we found in the lower abdomen. There were no adhesions about the cecum, which was apparently normal. I did not examine the sigmoid colon."

One month later the patient returned for treatment. His distress had returned after the operation, although not to the same degree. A strict dietetic and hygienic régime with colonic flushing gradually brought the patient into the best health he had enjoyed for several years. Strict injunctions against excessive indulgence in proteids, especially high meats and cheeses, were given, as experience had proved that a minimum of proteid was accompanied with less distress. With much proteid ingestion, indican always appeared in the urine and undigested meat fibers in the stools. When the diet was strictly followed, the stools lost their offensiveness, the gas diminished, and the number of Gram-positive bacilli decreased. Repeated rectal examinations were negative.

After three months of comparative freedom from trouble, the patient committed two dietetic indiscretions within twenty-four hours. Pain and distress in the hypogastrium followed, with temperature ranging from 99° to 104°, and a pulse of 100 to 120. His condition improved slowly for five days under treatment, when a sudden sharp pain followed an injection of water. The pain was so severe that morphine was given before the patient was seen. On examination there was considerable tenderness in the lower abdomen, some rigidity, but no cutaneous hyperesthesia. Rectal examination was negative. There was a little nausea, but no vomiting. A tumor mass could not be demonstrated at this time; there was some pain at the end of urination. On several occasions during the next few days the stools showed a little pus, mucus, and blood. Ten days later a mass had clearly defined itself in both iliac fossa, and could be detected per rectum as an irregular, elastic, bulging body. The patient's general appearance was decidedly septic, and he was removed to the hospital for operation. Leukocytes, 40,000.

From the clinical course it was assumed that the condition was a localized purulent peritonitis rising from the sigmoid. A chronic ulcerative process was excluded on account of the absence of blood and pus in the stools until the last few days. Perforation of a carcinoma was unlikely from the length of the clinical course

absence of blood, the absence of a tumor until a few days previous, (two years), the absence of signs of stenosis, the almost entire the character of the tumor, and its rapid development. By exclusion, diverticulitis of the sigmoid with the perforation of an infected diverticulum resulting in a pelvic abscess was considered probable.

Operation. An incision was made in the median line, and inasmuch as the most prominent part of the tumor mass seemed to the surgeon to be on the right side, a wide incision was made well to the right of the rectus muscle, and free drainage instituted. The case passed from observation, and death took place about six weeks after the operation. The autopsy report was as follows:

Operation about ten hours after death. Skin very pale. Slight rigor mortis. Peripheral lymph glands not enlarged. Two operative wounds on abdomen—one in mid-line about 10 cm. long; one over appendix about 7.5 cm. long. The latter drained. Subcutaneous fat well developed.

Upon opening the peritoneal cavity it was seen that the omentum was adherent over the whole anterior surface of the peritoneum, and less extensively to the intestinal coils. It was rolled into a mass in the left hypochondrium.

The intestinal coils were more or less generally adherent by old fibrous adhesions, and were also adherent to the parietal peritoneum in the right flank. The coils of the ileum were closely adherent to one another, and the lower ones were also adherent to the sigmoid by old and recent adhesions.

The cecum was bound down by adhesions, most of them recent, the results of an operation done some days before to drain the cecal and pelvic regions. There was no pus in the pelvis or about the cecum. The appendix had been removed.

The intestines were carefully dissected out and no other process than an adhesive fibrosis was encountered until the lower loops of the small intestines were dissected away from the sigmoid, just at the brim of the pelvis. At this point an abscess cavity situated under the sigmoid and walled off by the small intestine was opened.

As the large intestine was removed, pus was again encountered to the left and beneath the descending colon and sigmoid. This suppurative process could be traced to the abscess cavity mentioned above and to a larger one to the left of it, in the postintestinal tissues and just at the brim of the pelvis. The process had apparently also extended down (judging from the adhesions), but this extension had been limited by adhesions in the pelvis. Extending up along and behind the descending colon the line of extension of suppuration could be followed to the spleen, which was half enclosed in an abscess cavity.

An incision through the diaphragm from below and exploration of the left pleural cavity occasioned a gush of foul-smelling, greenish-yellow, thin pus to the amount of about 500 c.c.

The thorax was then opened and examined. The left lung was partially compressed by an empyema that extended from the diaphragm to near the apex. The pleura was covered by a greenish-yellow fibrinopurulent exudate. The left apex was the seat of a large healed scar, and scattered about on the pleura and in the pulmonary tissue were fibroid and calcareous obsolescent tubercles.

The right lung was also the seat of a healed tuberculous process, the pleura being thickly studded with obsolescent tubercles, especially at the junction, posteriorly, of the interlobar clefts.

The heart was pale but otherwise normal.

The liver was small, pale, and exceedingly soft.

The kidneys were very large and pale. The capsules stripped, leaving an untorn surface. Section of the organ showed the cortex enlarged, pale, and granular in appearance, with little line of demarcation between it and the medulla.

The adrenals were normal.

The pancreas was normal.

The spleen was of normal size, but soft, and the seat of an acute suppurative perisplenitis.

When the intestines were opened it was seen that the small intestine was atrophic, the walls thinned, the rugæ not prominent, and the mucosa thinned. From the cecum down, the submucosa and the muscularis became gradually thickened until, in the vicinity of the brim of the pelvis, the wall was from two to five millimeters thick, and perisigmoidal and rectal tissues were generally firm and infiltrated.

The sigmoid and rectum showed accentuated diverticular pouches, all of which had thinned distal ends and mouths about which the submucosa and the muscularis were very thick and edematous (Fig. 4). Extending from the distal end of one of these pouches was a sinus, surrounded by thickened fibroid walls, that communicated with the larger postcolonic abscess cavity. From this pouch or diverticulum the suppurative process had apparently originated. The tops of the folds of the descending colon and sigmoid showed numbers of small areas having somewhat the appearance of shallow ulcers. These areas measured not more than one by two millimeters. They had slightly thickened, raised, and rounded margins, and gray bases.

Anatomical Diagnosis. Anemia; catarrhal sigmoiditis and proctitis; diverticulum formation; chronic perforative diverticulitis; suppurative perisigmoiditis, pericolitis and perisplenitis; empyema; parenchymatous degeneration of the kidneys; fatty degeneration of the liver; obsolescent, calcareous pulmonary tuberculosis; left apical scar; general organized peritoneal adhesions; operative wounds.

The striking clinical features of this case are the absence of constipation, the indefiniteness of the bowel disturbance, the repeated

presence of large quantities of indican in the urine, the unfavorable influence of a proteid diet, the persistent rapidity of the pulse in all of his attacks, the distress at the end of urination, the fact that perforation evidently followed distention of the sigmoid with water, the fact that a surgeon of high diagnostic ability operated for what seemed to be an acute attack of appendicitis, the interesting distri-

FIG. 4.—Photograph of the sigmoid of Case I, to show the site of the perforated diverticulum and the mouths of other small ones (*X*).

bution of the tumor mass, which extended from the left iliac fossa down into the *cul-de-sac* and up into the right iliac fossa, where it reached its greatest prominence, and that this led a surgeon of large experience, after making a median exploratory incision, to drain the apex of the abscess in the right iliac fossa, with the belief that the seat of the trouble was there.

CASE II.—The following case was seen with Dr. Schleier, of Omaha. J. S., male, aged eighty-three years. The patient entered St. Joseph's Hospital December 9, 1908, in a semicomatose condition. No history could be obtained except that three days before admission he had had some trouble in passing urine and had, therefore, been catheterized. Subsequent urination had been free and the urine bloody.

General examination proved negative, except for a pulmonary emphysema and a well-developed arteriosclerosis. The abdomen was distended. Peristaltic waves could be seen starting in the region of the cecum, disappearing in the right hypochondrium, and again appearing at the median line, thence proceeding to the left and downward to the hypogastrium. There was no abdominal rigidity. There was tenderness posteriorly over both kidneys, with an area of renal hyperesthesia on both sides above the crest of the ilium. There was no abdominal tenderness. The bedsheets were stained with bloody urine; a specimen could not be obtained by catheterization. The breath was not urinous.

Rectal Examination. The rectum was ballooned, the prostate moderately enlarged, but not tender. No tumor mass was palpable.

Clinical Diagnosis. Chronic intestinal obstruction, probably due to stricture, cause unknown. Without a urine examination a chronic intestinal toxemia was suggested as a possible cause of the hematuria. Colotomy was advised and performed by Dr. Schleier. Death occurred about eighteen hours after operation.

Autopsy was performed eight hours after death. The patient was a somewhat emaciated elderly man. There was moderate edema and posterior congestion of both lungs, and marked senile emphysema. The heart was normal in size; there was tremendous calcification of the mitral valves and complete calcification of certain areas in the coronary arteries; there was also calcification of the aortic valves, and marked atheroma of the aorta throughout its entire length. The celiac axis was almost completely obliterated. The large intestine was dilated throughout its entire length; the hepatic flexure was adherent beneath and covered by the liver, a condition which explains why peristalsis could not be seen in this region. A colotomy wound was observed at lower part of descending colon. There was a stricture at the junction of the middle and lower third of the sigmoid. Numerous false diverticula were present in the area of the stricture, which was about one and one-half inches long (Fig. 5). There was a slight perisigmoiditis at the site of the stricture. Diverticula were numerous for a distance of a foot or two above the stricture. They varied in depth; their mouths were generally wide, and they entered the appendices epiploicæ. There was atrophy of the walls of the colon and of the small intestine. The kidneys were normal in size, the capsules stripped easily, and the surface was slightly granular. The cortex was atrophic, but there

were no gross signs of inflammation. The pelvis and both kidneys were markedly injected and covered with a dirty, sanguinopurulent exudate. The ureters were distended and similarly inflamed. They were so red and injected as to look like veins from the outside. The bladder wall was thickened and markedly injected. The bladder contained a small amount of bloody urine which showed pus cells and desquamated epithelium in large amounts. There were microscopic clumps of a dozen or more epithelial cells together,

FIG. 5.—Photograph of the sigmoid of Case II, to show diverticula in the portion involved in the stricture.

showing that desquamation *en bloc* of areas of bladder, ureteral, and pelvic epithelium had occurred. The prostate was hypertrophied; there was marked peripheral arteriosclerosis, with extensive deposits of lime salts in the vessel walls. The pathological diagnosis was emphysema and passive congestion of the lungs; coronary arteriosclerosis and calcareous deposits in mitral and aortic valves; general arteriosclerosis; passive congestion of liver and spleen; senile atrophy of spleen; chronic intestinal obstruction

due to benign cicatricial stenosis of sigmoid; false diverticula of the descending colon and sigmoid, especially at area of stricture; dilatation and atrophy of the colon; hypertrophy of the prostate; ascending cysto-uretero-pyelitis.

The intimate relation of the numerous false diverticula to the stenosed areas in this case makes probable their pathogenic significance in benign cicatricial stenosis of the sigmoid.

CASE III—S. B., farmer, aged fifty years, entered St. Joseph's Hospital January 4, 1909. The patient was in a stuporous condition, and the following history was obtained with difficulty. The duration of illness was indefinite and onset had been gradual. The patient complained of intermittent abdominal pain, which was described as "sore, colicky," of belching, headache, weakness, and of constipation. His bowels had not moved at all for three days, and not well for several weeks previously. No pulmonary, cardiac, or renal symptoms could be elicited. The past history was negative except for indefinite trouble with the lower bowel, and constipation. Later, a history was obtained of a disturbance, nine months previous, in the lower abdomen, which lasted several weeks, and was characterized by colicky pains and distress in the median line and to the left, alternating diarrhea and constipation with mucus in the stools, fever, and soreness in the left lower quadrant. The condition of the patient when first seen was as follows. He was an emaciated, medium-sized man, stupid in appearance, and apparently of some sixty years of age. He lay quietly in bed, yawned continuously, his eyes were half closed, and he paid but little attention to what was going on around him. It was necessary to speak loudly and prod him to obtain answers to questions. Both lids were equally ptotic, but he could raise them with the assistance of the frontal muscles to the level of the iris. The pupils were equally contracted, and reacted slightly to light and distance. The breath was foul, and the teeth poor. Vision seemed good. There was no asymmetry in muscle power. The patient could recognize a watch tick for a distance of only about an inch from each ear. Examination of the neck, lungs, and heart was negative. The abdomen was soft and not especially tympanitic. The liver and spleen were negative. Peristaltic waves occasionally could be seen traversing the hypogastric region from right to left. They were about two to three inches in breadth and were accompanied by signs of distress and rubbing of the abdomen. Rectal examination, save for "ballooning" was negative. No tumor mass was palpable. The urine was highly concentrated. It contained indican and urobilinogen (Ehrlich's aldehyde reaction) in large amounts, a trace of albumin with occasional hyalin, and granular casts. The superficial reflexes were markedly diminished, and the deep reflexes were faint or absent, but symmetrically so. The temperature was 101°, and the pulse 70. An injection of warm olive oil was ordered, with a

mercurial and saline, which brought away a large amount of fecal material. The mental condition brightened for several days. Then relapses occurred at varying intervals with conditions similar to those described. The trouble always could be relieved by a rectal injection of one to two quarts of water. A diagnosis of chronic intestinal obstruction, of unknown cause, in the region of the sigmoid, was made at this time. On January 25, 1909, the patient developed a double lobar pneumonia, which lasted ten days. During this time injections of water were given each day.

On February 28, 1909, another attack of pneumonia occurred which produced stupor and was accompanied by signs of intestinal obstruction, *e. g.*, peristalsis and obstipation, which were relieved only by repeated injections of both oil and water. A large sacral decubitus four inches in diameter developed at this time. Several attacks of obstruction followed, which were easily remedied by injections. During intervals of from one week to ten days the patient had normal although constipated stools.

It was clear that an intermittent intestinal obstruction existed. This must have been caused by a mechanical factor prevailing only at intervals. The occasional copious and well-formed movements spoke against stricture. The constant absence of blood in the stools, and of a tumor mass, spoke against malignant disease. There was no evidence of hernia. Of causes that could produce such a frequent recurrence of obstruction, followed by free intervals, only two could well be assumed. The first was adhesions. But a band could hardly produce this picture by mere constriction. Traction of an adhesion on a loop of intestine might cause kinking when influenced by changes in intra-intestinal pressure, and by variations in the position and caliber of the gut, both of which factors are, to a certain extent, dependent on the intestinal contents. The second possible cause was intermittent volvulus upon the basis of cicatrization and retraction of the mesosigmoid forming an axis on which rotation of the sigmoid might occur. Such a condition might have accounted for an intermittent intestinal obstruction, which was not relieved by catharsis, but by rectal injections. A history of intermittent intestinal trouble with fever, pain, and diarrhea, with the presence of blood the year before, suggested the possibility of such a condition.

Operation was advised on the basis of a diagnosis of intermittent chronic intestinal obstruction, due to volvulus or to adhesions. An old inflammatory process resulting from a diverticulitis was suggested as a possible *causa morbi*.

The operation was performed by Dr. C. C. Allison. An incision was made to the left of the median line. The upper and lower segments of the sigmoid were found separated by about one inch of scarred mesosigmoid. There were slight adhesions running from the origin of the sigmoid to the parietal peritoneum. Several

diverticula were seen close to the mesenteric attachment in the vicinity of the retracted mesosigmoid. It could not be demonstrated without opening the gut, that the cicatrization was secondary to a diverticulitis, and so, although the existence of the diverticula may have been a coincidence, yet inference to the contrary is permissible. The upper limb of the sigmoid was freed from adhesions and stitched to the parietal peritoneum higher up. A complete recovery took place.

Case IV.—N. A. C., male, aged seventy-five years. The case was a coroner's autopsy. The anatomical diagnosis was: Myomalacia cordis; sclerosis and obliteration of the right coronary artery; general arteriosclerosis; passive congestion of liver, spleen, and kidneys; chronic interstitial nephritis; pulmonary emphysema. Several false diverticula of the sigmoid were found entering the appendices epiploicæ.

Case V.—E. R., male, the anatomical diagnosis was: Healed tuberculosis of both apices; chronic bilateral pleural adhesions; dilatation and hypertrophy of the heart; myomalacia cordis with cardiac aneurysm; passive congestion of lungs, liver, kidney, and

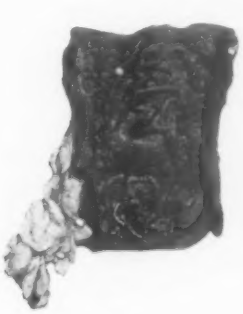


Fig. 6

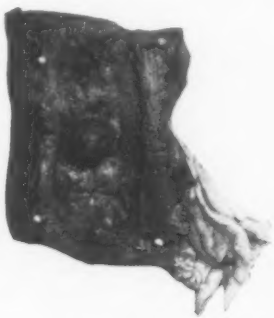


Fig. 7

Fig. 6.—Photograph of one of the diverticula in the sigmoid of Case V.

Fig. 7.—Photograph of the same diverticulum in Case V, showing the inspissated feces contained in it.

spleen; cholelithiasis; arteriosclerotic kidney; diverticula of the sigmoid and descending colon. The diverticula were located in the vicinity of the mesenteric attachment. Their walls were extremely thin, and they contained inspissated feces (Figs. 6 and 7).

Case VI.—The case was a coroner's autopsy. The subject was an elderly obese male, of unknown age. The anatomical diagnosis was: Edema of lower extremities; passive congestion and edema of the lungs; hypertrophy and dilatation of the heart, especially of the left ventricle; degeneration of the myocardium; passive congestion of the liver, spleen, and kidneys; chronic interstitial

nephritis; false diverticula of the sigmoid. The sigmoid was surrounded with a large amount of fat, and the appendices epiploicæ consisted of large fatty masses. Six false diverticula could be seen penetrating these fatty masses (Fig. 8).

FIG. 8.—Photograph of a portion of the sigmoid of Case VI, to show diverticula.

DIAGNOSIS. It can be easily seen, from the brief *resume* of the pathology of diverticulum disease, that the symptoms may be manifold and difficult of interpretation. The diagnosis is seldom easy, rarely certain, and often impossible. Franke was able to collect from the literature only 2 cases in which the diagnosis had been made. To these he added a case of his own. Graham recently suggested the diagnosis in one of Mayo's cases. To appreciate the possibilities is a long step in the direction of a diagnosis, which must be made largely by exclusion. The most easily recognizable condition is that of the so-called "left-sided appendicitis." There is pain, gastric disturbances, tenderness, and rigidity in the left lower quadrant, with temperature and leukocytosis; abscess formation often appears later. Another class of cases that is moderately

easy of recognition is that of chronic hyperplastic sigmoiditis. This has been treated as a disease entity by Ewald, Boaz, Rosenheim, and others, but it is possible that diverticulitis may be found a basis for it. In this complex there is always intermittent pain, disturbed function associated with irregularity of bowel action, gas, and often spastic stools in which there is frequently an increase in mucus and occasionally a trace of blood. Physical examination discloses a tender sausage-shaped tumor which may be either adherent or freely movable, and which varies in size from time to time. Infiltration may be felt occasionally per rectum. Sigmoidoscopy shows a swollen, reddened, rugous mucosa, and often narrowing of the lumen of the gut. Obstructive symptoms may be present, but are not so common as in cancer, from which condition it must be differentiated. In carcinoma the clinical course is more progressive and definite. Its duration is limited; ulceration with blood in the feces is the rule, and when blood once appears it is usually quite constant. The tumor is harder, more likely to be movable, definitely circumscribed, constantly increases in size, and is not elongated. Bladder symptoms are apt to be frequent in diverticulitis, as the bladder is often involved in the inflammatory process. Cripps states that vesicosigmoidal fistula is more commonly inflammatory than malignant (45 out of 63 cases). If such be the case, many of them may be the result of a diverticulitis. Disturbed bowel function is constant. Many cases record constipation as a more or less pronounced prodromal symptom. Pelvic abscess, tuboövarian abscess, mucous colitis, amebic colitis, chronic proctitis, tuberculosis and syphilis of the sigmoid, psoas disease, retroperitoneal abscess in the left flank from other causes, etc., must, in addition to the conditions already mentioned, be differentiated.

TREATMENT.—The treatment must be surgical in many cases. The prognosis is often a question of technical efficiency. It is certain that, as in appendicitis, many of the cases will recover without operative interference. Just what are the indications for resection in hyperplastic sigmoiditis is a question for future determination. Abscesses and vesicosigmoidal fistulæ should always have the advantage of surgical treatment. As knowledge of the morbid anatomy of false diverticula increases, as cases are recognized, reported, and treated, as methods of surgical procedure become crystallized, facts will be obtained for reliable diagnosis and prognosis.

LITERATURE.

Fischer. Jour. Exper. Med., 1900, v, 33.
Beer. AMER. JOUR. MED. SCI., 1904, cxxviii, 135.
Telling. Brit. Med. Jour., October 31, 1908, 2496.
Franke. Deut. med. Woch., 1909, xxxv, 98.
Eisendrath. Arch. Diag., October, 1909.
Giffin and Wilson. AMER. JOUR. MED. SCI., 1909, cxxxviii, 661.

PERFORATING ULCER OF THE DUODENUM: DEATH FROM PULMONARY THROMBUS.[1]

By Geo. Tully Vaughan, M.D.,

PROFESSOR OF SURGERY, GEORGETOWN UNIVERSITY; CHIEF SURGEON TO THE GEORGETOWN UNIVERSITY HOSPITAL; SURGEON TO THE EMERGENCY HOSPITAL; CONSULTING SURGEON TO THE GOVERNMENT HOSPITAL FOR THE INSANE, WASHINGTON, D. C.

This patient, a young man, aged twenty-nine years, was sent to me by Dr. W. C. Ford, of Woodstock. He gave the following history: For some years he had suffered at times with pain in the stomach, and what he called "indigestion." Two days before his admission to Georgetown University Hospital he was taken in the morning with griping in the bowels, which was relieved by going to stool. About 5 P.M., of the same day he was suddenly taken with severe pain in the abdomen, chiefly in the epigastrium, but extending below the level of the navel and upward on the right side to the chest and shoulder. He vomited several times a prune-juice looking material, and later on, still darker colored material. No chills.

On admission to hospital, forty-eight hours later, his pulse was 130, temperature 99°, abdomen slightly swollen, tympanitic, rigid, and tender, especially over the gall-bladder region, slightly over the appendix. In making the diagnosis the following conditions were considered in the order of probability: Perforating ulcer of the stomach, bowel, or gall-bladder, or appendicitis, and immediate operation was advised and was done. The incision was made through the right rectus muscle, and the region of the appendix was examined and exonerated. On passing the fingers upward, adhesions were felt and a turbid greenish fluid began to escape. The incision was prolonged upward, and a patch of lymph was seen on the anterior surface of the pylorus and first portion of the duodenum, with a hole about $\frac{1}{5}$ of an inch (1 cm.) in diameter in the centre of it, leading into the bowel, it was thought, from which the greenish-colored fluid was escaping. It was closed at once with fine silk sutures. Plastic lymph was found on the gall-bladder, liver, stomach, intestines, and anterior abdominal wall—extensive peritonitis existing and an abscess containing thick pus was found behind and below the right lobe of the liver. A rubber drainage tube wrapped in iodoform gauze was placed in the abscess cavity; the abdomen was wiped out with gauze, and closed around the drainage tube. Pulse 140 during and at the close of the operation. Salt solution by rectum, six ounces every hour, was ordered. From this time the patient steadily improved, and at the end of the seventh day his pulse and temperature were normal, the tube was draining freely, and his general condition was satisfactory. On the tenth day

[1] Read at the Virginia Medical Society in Norfolk, Va., October, 1910.

the temperature in the evening rose to 101°, pulse to 100, but the patient slept pretty well. Next morning the temperature was 99°, pulse 90, and he said that he did not feel so well, had some pain in the right side, and preferred to lie on his back, as it hurt him to turn. No trouble with respiration. About 4 P.M., while using the bedpan after an enema, he suddenly complained of difficulty in breathing, pulse became very rapid, his face and chest became cyanotic, he insisted on being raised up, but in a few minutes fell back dead—the entire attack not lasting over fifteen minutes.

This result was unfortunate and disappointing to the physician and to the friends of the patient—to have him snatched from the jaws of death and still lose his life, as if a man rescued from drowning should be suddenly struck dead by lightning—it was most discouraging. The cause of his death could hardly have been anything else than a cardiac or pulmonary thrombus, and this was probably caused by the infection of the abdominal cavity.

Pulmonary thrombus or embolism is said to be not very common. Albanus found that it followed 2 per cent. of abdominal operations. It seems to be more common after gynecological than general surgical laparotomies. Burkhard gives in 236 operations for uterine fibroids 12 cases of embolism (Keen's *Surgery*). While pulmonary and other forms of embolism are usually preceded by some kind of an infection, yet there are many cases in which the infection is not recognized or does not exist. Rough handling, bruising of the veins, has been given as a cause for embolism. I have seen many cases of embolism or thrombosis of the iliac and femoral veins following removal of the appendix, of the uterus, and other surgical operations in the abdomen, but I have seen only three cases of pulmonary embolism, and two of them were fatal. The first occurred in a boy, aged fourteen years, suffering from an infected gunshot wound of the knee-joint, the ball lodging in the joint, from which it was removed by incision. The symptoms came on five days after the receipt of the wound, although he had vomited the day before, and rested poorly that night. On the morning of the fifth day his pulse was 80, but very feeble, heart sounds almost imperceptible, skin cyanotic and cool all over body, no dyspnea, perfectly rational, and contented. Death occurred suddenly about half an hour after these symptoms appeared. The absence of distressing symptoms was probably due to the slow formation of the thrombus and the gradual arrest of the circulation.

The other case occurred in a woman, aged thirty-eight years, after an operation for double hernia. No infection existed so far as known, although the pulse ranged from 90 to 100 and temperature from 99° to 100°; but on the ninth day after operation the patient was taken suddenly with pain under the left breast, a feeling of oppression and difficulty in breathing, the pulse rose to 120, the heart sounds were weak and somewhat muffled, she had chilly

sensations, and vomited once. Nothing of note was heard over the lungs. These symptoms continued for about twenty-four hours, then gradually subsided, so that at the end of forty-eight hours the pulse, temperature, and general condition were nearly normal and recovery followed. In this case the embolus was probably small and was soon disposed of.

The treatment of thrombosis obviously consists in preventing infection and avoiding injury to vessels by rough handling during operations. The efficiency of the treatment by frequent change of position and short stay in bed after operations, in preventing thrombosis is still open to question.

A few surgeons have operated, opening the pulmonary artery and drawing out the thrombus, but so far without permanent success. Sievers and Trendelenburg, of Leipzig, have each operated on a case. Trendelenburg opened the pulmonary artery and removed a thrombus thirteen inches long, but the patient died thirty-seven hours later.

PURPURA HÆMORRHAGICA, WITH REPORT OF AN ATYPICAL CASE.

By A. C. Matthews, M.D.,

and

H. P. Carpenter, M.D.,

Assistant Physicians, Hudson River State Hospital, Poughkeepsie, N. Y.

Purpura is often looked upon as a disease, especially in that form known as the idiopathic variety, but it is not at all improbable that if the exact etiological factor in the so-called primary or idiopathic cases could be determined, we would have to consider this trouble as a symptom, rather than a disease. Although purpura has been carefully studied for many years by careful observers, we know very little of a definite nature about the causation of the primary cases. Dr. Osler has referred to purpura as "that obscure and interesting manifestation of which we know so much and at the same time so little." Histologically purpura used to be confused with the contagious fevers, especially the eruptions of scarlet fever and measles. It was not until the latter part of the seventeenth century that a clear description of this disease was given by Zeller. Before proceeding farther, it should be stated that the case to be reported belongs to the primary or so-called idiopathic purpura, which is not to be confused with the more ordinary type of secondary purpura occurring in such troubles as typhoid fever, nephritis, heart disease, lymphatic leukemia, pneumonia, arteriosclerosis,

alcoholic neuritis, cachectic states, as senility, iodine idiosyncrasies, and many others.

DIVISIONS AND FREQUENCY. It has been customary to divide idiopathic purpura into the mild, moderate, and severe grades, or purpura simplex, purpura rheumatica, and purpura hæmorrhagica. These types are frequently not sharply defined. That idiopathic purpura is a rare condition is shown by statistics.[1] At the Massachusetts General Hospital there were only 65 cases of idiopathic purpura among 155,884 patients during a certain period. At the Johns Hopkins Hospital, 41 cases were observed in 18,594 medical patients. At the Hamburg General Hospital there were 73 cases in forty-one years in a total of 100,000 patients. Some authorities believe the affection is not uncommon, but in such instances it is very probable that both the secondary and idiopathic forms are grouped together under one heading. Stelwagon states that the disease is not uncommon, then he proceeds under the heading of causal factors to mention such conditions as malaria, grip, profound anemia, syphilis, nephritis, etc., clearly showing that he has joined the two types under the common term purpura.

HISTORY OF THE CASE. The patient was an insane male, a case of dementia præcox, aged thirty-five years, whose physical condition previous to the onset of the trouble was at par so far as known, aside from an anemic appearance. During the night of June 21 he was taken suddenly ill with nausea and vomiting. There were two attacks. The vomitus contained nothing characteristic. There were a few food particles and much mucus at first, and the second vomitus consisted of a watery fluid, with some mucus. The following morning he ate no breakfast, but took some nourishment for dinner and supper. An examination of his physical condition revealed nothing abnormal. His stomach was not sensitive; bowels were regular; pulse and temperature normal. He complained of feeling a little weak in the morning. As he appeared to be in his usual health during the afternoon, he was allowed to dress and go about the ward. The following night he was again taken with three vomiting spells. He refused breakfast. There was no fever; pulse, 90. Examination showed some general tenderness over the stomach, which was produced probably by frequent retching. The patient was kept in bed, and showed no additional symptoms until the morning of June 24, when a profuse hemorrhage occurred from the bowels. Vomiting began immediately, and in the vomitus were observed brownish streaks (decomposed blood). The cause of the hemorrhage could not be ascertained. Leukemia, hemophilia, and purpura hæmorrhagica were considered. The blood examination eliminated leukemia. Hemophilia was discarded by a negative family history, a normal blood-clotting time,

[1] Osler's Modern Medicine.

the male sex, and development after puberty. For purpura hæmorrhagica there were no skin lesions.

The patient failed rapidly. During the following two weeks there was rapid emaciation, due to frequent and profuse hemorrhages from the bowels and stomach and inability to retain nourishment. Temperature was never above 100° F. Rectal feeding could not be resorted to, owing to his violent and resistive tendencies. Medicinal measures did not relieve the nausea, vomiting, and hemorrhages. Signs of collapse were apparent. Pallor was marked; expression anxious; temperature subnormal, with cold, perspiring skin; thready pulse, and great emaciation. July 6, while in this condition, there were presented the first signs which gave a clue to the affection we were dealing with. There appeared upon the extremities about the elbow- and knee-joints a few petechiæ, varying in size from a pin-point to an eighth of an inch in diameter. These gradually increased in number during the next few days, until there were two or three dozen spots on each extremity. Later, a few spots occurred upon the trunk. Now and then a small hematoma was seen. With the development of the skin lesions and the administration of special medicinal measures the constitutional symptoms began to abate. From this time on there was a slow, but steady improvement.

The interesting and unusual circumstance connected with this case is the fact that there were no evidences of any integumentary lesions until the constitutional symptoms had practically run their course and had almost caused a fatal termination. In the treatises on purpura to which I have had access, no mention is made of this peculiar course, but Osler's *Modern Medicine* speaks of purpura hæmorrhagica without skin lesions. To quote: "There are some cases of the hemorrhagic diathesis which present the clinical picture of purpura hæmorrhagica, except that cutaneous hemorrhages are absent. There is no hereditary or congenital tendency to hemorrhage, and hence they cannot be classed under hemophilia. In some chronic cases of this type the diagnosis is confirmed subsequently by the appearance of purpuric spots in the skin." Osler[2] has reported such a case, with recurring abdominal crises and bleeding at the nose for a year, before a purpuric rash appeared during an attack. J. W. Coe studied a patient in whom during a period of twenty-eight years there had been frequent attacks of epistaxis so severe that packing of the nostrils was often necessary. There was no history of bleeders in the family, and the subject had never had arthritis. Owen observed a case of this type in a girl, aged eleven years, who had hemorrhages for twelve months, first from the bowels, then the nostrils, and afterward from the

[2] Amer. Jour. Med. Sci., December, 1895.

left ear. There was no history of bleeders in her family, no arthritis, and no purpura.

ETIOLOGY. The exact cause of true or primary purpura and its mode of production are unknown. Symptomatic purpura may occur in many conditions, as I have already mentioned, but in the disease purpura the hemorrhagic tendency is not congenital or hereditary, in contrast to hemophilia. It is met with in both sexes and in all ages, but is most frequent in the second decade, and rare after fifty years. No one cause can at present be set down as essential in all cases. Microörganisms have been looked upon as causative in many of the more grave cases. At least three experimenters have succeeded in producing the malady by inoculating animals with pure cultures. Stelwagon believes that the causative factors may be divided, in general, into vasomotor, toxic, and infectious, but he includes the secondary with the idiopathic cases. In the case I have reported the symptoms would seem to indicate a toxicity of the intestinal tract, but whether this was an auto-intoxication starting from the intestinal tract or due to a toxic condition of the blood with elimination of certain poisonous products into the stomach and bowels can only be conjectured. The patient's anemic appearance might have suggested a deficiency in the hemoglobin and number of red cells as a possible etiological factor, but any anemic blood condition which might have existed prior to the onset of the trouble could not have been very pronounced, for after several large hemorrhages had occurred, the red blood cells numbered 3,300,000 per cubic millimeter.

PATHOGENESIS. In considering the pathogenesis of purpura, we must necessarily turn our attention to the study of the condition of the blood and bloodvessels. As the question is still unsettled, it would seem important to give a few of the more recent views concerning its production and the changes accompanying it. It is certainly well established now that the bloodvessels are concerned in the production of purpura, yet the changes in the vessels may be induced secondarily by the alterations in the blood composition. The changes found in the bloodvessels at autopsy are not constant. Riehl describes the presence of localized degeneration of the vessels in all cases of purpura. Adami, in his recent work on *Pathology*, speaks of a fatty hyaline degeneration occurring in the walls of the smaller vessels in all cases except the neuropathic variety. Von Kogerer claims that a careful search would always reveal the presence of thrombi due to vascular degeneration. According to this theory the disease of the vessels leads to thrombosis and this in turn leads to the hemorrhages. The way by which the blood cells leave the vessels is not known. Many pathologists have examined the purpuric spots without finding any breaks in the vascular walls. Clinicians object to the theory of primary vascular degeneration, for it is difficult to see how the hemorrhages can be so widespread

over the surface of the body and develop with such rapidity. Also the tendency to hemorrhage is transitory, and one cannot readily conceive of so severe a disease of the bloodvessels being such a transient affair. Flexner discovered a substance in snake venom which when injected in laboratory animals possessed the property of destroying endothelium and producing multiple hemorrhages in a few minutes. Gay and Southard also report experiments in which the lining epithelium of the capillaries was so changed that focal hemorrhages occurred in as short a time as four minutes. These experiments certainly suggest the possibility that in purpura there is a substance present in the blood which produces an endotheliolysis with focal hemorrhages.

We will now discuss briefly the changes that occur in the blood itself in purpura. Dennes, a Belgium pathologist, and Hayem, in 1890, noticed the marked diminution of blood platelets in purpura. Recent investigators have confirmed these findings, and even say that it is characteristic. Normally, the platelets number from 200,000 to 600,000 per cubic millimeter. In purpura they range from 10,000 to 42,000 or higher, but in practically all cases they are markedly diminished. Hayem has described another change in the blood in purpura. Normally, the blood clot quickly contracts and expresses serum. The clot in purpura hæmorrhagica does not retract, and there is no extrusion of serum. Others have confirmed this finding, and experiments have been performed to ascertain the behavior of blood from which the platelets have been removed. It was found that when the platelets were filtered out the blood clotted slowly, and the clot did not contract and no serum appeared. It seems quite natural, therefore, to ascribe the contraction of the clot with expression of serum to the presence of the blood platelets which we find so diminished in purpura hæmorrhagica. Albumose injected into the circulation of animals diminishes the number of platelets, and Hayem produced a condition analogous to purpura by injecting the serum of an animal of another species. The platelets collected in masses forming thrombi, leaving very few in the circulatory blood. He concludes that the diminution is due to their destruction, which may be brought about by the introduction of toxins from the digestive tract. This same view of auto-intoxication is also held by other prominent writers. It seems quite probable that in cachectic states, such as leukemia, nephritis, carcinoma, etc., with purpura that the toxic substances formed are likely to produce profound changes in the blood, which lead to rapid disintegration of the vessel walls or to the slower hyaline or fatty degeneration. This, however, is not the idiopathic variety. Undoubtedly there is a combination of factors at work in the production of purpura. As a basis of the condition we have a change in the composition of the blood causing a more or less marked disintegration of the lining epithelium of the vessels, which allows the blood to pass through

into the tissues giving the hemorrhagic spots. One of these very prominent alterations we have seen in the marked diminution of the blood platelets. Whether this alteration is dependent upon an obscure infection or upon an autointoxication is problematic. It would be interesting to determine in cases of autointoxication if there is a diminution in the number of blood platelets, and if the blood clot lacks an inclination to contract quickly and to exude serum.

It is unfortunate that the case we have here reported was not worked up from the laboratory standpoint at the early part of the disease and later at the height of the process. The patient was well along in convalescence when the blood examination was made. There was a moderate anemia, the red cells numbering 3,300,000 per cubic millimeter. The leukocytes were practically normal, numbering 7300. This finding rather discourages the idea of infection in this case, unless we were too late to observe a leukocytosis. The blood platelets as we are led to expect were markedly diminished, numbering only 18,700 per cubic millimeter, instead of from 200,000 to 600,000 in the normal blood. We were unable to verify the non-contraction of the clot and failure to express serum, but we must consider that the case was far along in convalescence at the time of the examination. The urinary examination at the time of the patient's illness was negative aside from a large increase in indican which so often accompanies auto-intoxication.

TREATMENT. The successful treatment of any disease depends upon getting at the etiological factors. Of course, in secondary purpura we direct our attention to the conditions under which they arise. Whether or not we are dealing with the idiopathic or secondary forms, the patient should be kept in bed to insure absolute rest. The mildest types may not require this. Among the various agents used should be mentioned sulphuric acid, ergot, turpentine, gallic acid. Opinions vary as to their therapeutic value. Osler favors oil of turpentine in 10 to 15 mm. doses, three or four times a day. It is known to be a hemostatic, but owing to its irritating tendency and consequent liability to cause inflammation of the kidneys it should be used with care. Stelwagon has met with success in the grave forms with ergot given by subcutaneous injections. Adrenalin has been very beneficial in hemorrhages from the mouth and stomach. If used for intestinal hemorrhage it should be given hypodermically, as it is decomposed in the stomach. Owing to the persistent nausea and vomiting in our subject at the height of the trouble, nothing could be retained on the stomach, not even cracked ice. Wright has advised for the hemorrhages a trial of calcium chloride in 20-grain doses three times daily, basing its probable value upon its service in lessened blood coagulability, which from his investigations seemed to be the direct etiological factor. The remedy should not be given, however, for more than

several days, as its continued use finally diminishes the coagulability. Pratt, reporting in Osler's *Modern Medicine*, said that in some of their severe cases the use of calcium salts was of no benefit. On July 6 the case here reported was a very sick man. Vomiting continued, with occasional hemorrhages. This day he was placed upon 20-grain doses of calcium chloride, three times daily. From this time on recovery, though slow, was steady. There were no more nausea, vomiting, or hemorrhages. After four days he was given a rest from medication for three days, and then placed upon calcium lactate for one week. Tonics, a liberal diet, and iron aided the convalescence.

Just how much credit should be given the calcium chloride and lactate administered in this case cannot be stated with any degree of certainty. As before mentioned, Dr. Pratt has not met with success in using calcium chloride in some severe cases; others report more favorably.

We are very grateful to Dr. Chas. W. Pilgrim, superintendent of the Hudson River State Hospital, where this case was observed, for granting permission for its publication.

NEOPLASMS WITHIN THE SKULL: THEIR EARLY DIAGNOSIS AND SOLE TREATMENT.

By Tom A. Williams, M.B., C.M.,

CORRESPONDING MEMBER SOC. DE PSYCHOL. ET SOC. DE NEUROL. DE PARIS; MEMBER ASSOCIATE SOC. MED. MENT. CLIN., ETC.; NEUROLOGIST TO EPIPHANY FREE DISPENSARY, WASHINGTON, D. C.

The treatment of intracranial tumors is at present in the same false position as was that of appendicitis fifteen years ago. At one time all cases, and even now most of them, die without receiving any real treatment; for the giving of potassium iodide is merely ritualistic. No case is recorded in which a postmortem has proved that a non-specific growth had been cured by drugs. As short of surgical removal, only death is to be expected, the term "expectant" applied to other treatment is a misnomer unless the expectancy refers to the patient's certain death. Temporary improvement, whether spontaneous or from narcotics or potassium iodide, does not justify postponement of the only real treatment. It would be as reasonable to postpone an appendectomy because of the comfort a victim might derive from opium.

Even in most of the cases which do come to operation, careful clinical investigation reveals an unequivocal history of intracranial disease, often of several years' duration. It is our duty to find out

and enunciate the earliest signs which point to intracranial neoplasms, in order that we may bring to the patient, before he is hopelessly moribund or permanently damaged, that relief which can be obtained in only one way. I refer to surgical removal of the growth.

This is my own firm opinion, but the words are a condensation of those of Sir Victor Horsley, delivered in October, 1910, to the German Society of Neurology at Berlin. I am the more impelled to quote them here because of the recalcitrance which I have encountered regarding my urgent advice to operate upon a number of cases about which I have been consulted in Washington. In no single instance has my advice been followed, and the patients have all died, with the exception of one seen only a few weeks ago, and of another of whom I have lost sight.

Before relating a few of these, it is good to emphasize the symptoms which should create suspicion of a cerebral tumor. Fine focal epilepsy is the most striking of these. Bravais-Jacksonian attacks are rarely due to other than local irritation. The diagnosis of epilepsy and the exhibition of bromides in such cases denotes an ignorance utterly injustifiable at this day. Such a case should be at once examined by someone versed in neurological technique. By refined measures of diagnosis, signs may be elicited by which an interpretation can be made, and a surgeon can then be called in at a time when a growth can be removed with certainty and without danger. The same remarks apply not only to focal epilepsy, but to any focal symptom at all. Thus, slowly progressive or recrudescent paralysis strongly indicates neoplasm, when granulomas are excluded. Slight losses of the attitude or muscle sense in one hand are similarly significant. So also is diminution of the other sensibilities. General or partial dystaxia or dysergia should always be investigated from this point of view. Slight differences or irritations of the special senses are the earliest symptoms of some cases. Aphasia of different types may be the first symptom. All these, in the early stages, have great focalizing significance. But it is not sufficiently realized that certain forms of mental dulness, of amnesia, of emotionalism, and changes in character are of localizing import as regards the destructive or irritative effects of a tumor of the frontal lobe.

Now, when one of these symptoms is found and a neoplasm is suspected it has been the procedure to wait for signs of cerebral compression before resorting to operation. The absurdity of this antediluvian attitude should need neither indication nor insistence, but that both are required is shown by my experience in Washington. An analogy will make it clear. Suppose that a surgeon, before removing a diseased appendix, were to wait for perforation, peritonitis, and abscess formation; does any one suppose that the mortality from appendicitis operations would be negligible, as at present? Would not such a diagnostician be a laughing stock? Again,

if the removal of carcinomata of the breast was always delayed until glandular involvement assured the diagnosis, would not their fatality approach 100 per cent? I doubt if the surgeon who awaited this kind of certainty of diagnosis would ever find a case to operate upon.

Now, the neoplasms found within the cranium are often benign, and none have the malignancy of mammary carcinoma. Moreover, many of them are diagnosticable, while very small, on account of the functional responsiveness of the tissue they invade. Hence their operability, technical difficulties apart, should be more hopeful than that of cancer of the breast, bowel, or uterus. Again, the subarachnoid sac is a less delicate structure than is the peritoneum, and the brain is less easily traumatized than are some of the structures of the abdomen. Hence the technical difficulties are largely mechanical, and, indeed, many of them are already overcome by the labors of Horsley, Krause, Cushing, and others.

Hence exploratory operations within the cranium are no more dangerous and no less justifiable than they are within the abdomen, always provided that sufficiently precise indications can be furnished the surgeon by the diagnostician. These are now available, and have been laid down by myself and others following such authorities as Bruns, Oppenheim, Duret, Horsley, Collier, Allan Starr, Mills, Spiller, Cushing, and others. It is only by their utilization that it is possible to detect an intracranial growth before an extensive destruction of tissue has occurred, and it is only then that an operation can be successful, in so far as complete restoration of function is concerned, and that is what should be aimed at by every physician. Even when the opportunity for intervention is too late to restore function, however, a proper operation can nearly always prolong or even save life.

The corollary of these propositions is that drug or expectant treatment is utterly unjustifiable, for the only eventuality to be expected from such measures is the patient's incapacity and death.

Case I.—A woman, aged about fifty years, seen with Dr. Gerry Morgan, who suspected some cerebral trouble because of his failure to prevent recurrent vomiting. There had been severe headache, staggering gait, and fainting spells. I found the woman in bed, somewhat dazed. Memory was much impaired, and she had to search for her words. There was no word deafness, nor aphasia, properly speaking, but incoherence had appeared now and then. I found a complete left lateral homonymous hemianopsia, but no contraction of the color fields other than this. The pupils reacted to light. There was marked edema of both optic papillæ. There was slight nystagmus with the lateral movement of the eyeballs. The attitude sense of the right hand was impaired. There was a right facial paresis, but no other motor loss. The gait was not cerebellar, and hardly uncertain. The plantar reflexes were in flexion. The

Achilles reflexes were diminished, the right being almost absent. The right patellar reflex was weaker than the left. The brachial reflexes were absent, as was the right abdominal reflex.

On account of the headaches, projectile vomiting, amnesia, difficulty of speech, and choking of the optic disks, I believed that there was a growth, and that it was in the right occipitotemporal region on account of the left lateral homonymous hemianopsia. I advised operation, to be preceded by a few days further observation and study to define the locality still more precisely. But the next day I was suddenly again called to the case. We found the woman curled upon the left side, stuporous, and vomiting. On examination, the plantar reflex was now in extension. The pupils were more contracted, but reacted fairly well. There was no further modification of sensibility, as far as could be found. We had taken with us Dr. Wellington, realizing in advance the need for urgent operation. After consultation, we advised removal to a hospital at once, and operation; but we did not decide whether to decompress in the subtemporal region or to uncover the presumed seat of the growth. However, the decision was never needed, for the family summoned a former attendant, who in turn called in two other consultants, whom he well knew would discountenance trephining, and the patient was allowed to die, which she did shortly afterward.

Case II.—Referred for opinion and care by Dr. Carl Henning. Complains of feelings in the head, "as if twisting around," for over three years. The vision has been much dimmed. She has had convulsions, which she cannot describe, severe frontal headaches, stiffness and pain at the top of neck behind, slight nausea, occasional dizziness. On examination, I found a commencing atrophy of the optic disk, with a swelling of about one dioptre. Great asthenia. An uncertain, staggering gait, not cerebellar in type. Great slowness of thought and speech, with incapacity to remember details clearly. No apraxia could be elicited. Rotation tests were negative. The deep reflexes were exaggerated, but plantar flexion was prompt and complete. The smell was defective in the left nostril, and the sound of the Weber tuning-fork test was lateralized to the left. No other sensory modifications could be ascertained.

Without observation or actual description of the convulsions, localization could not be precise; but that there is a growth present needs no argument, and that it is in a frontal lobe is to be presumed from the convulsions, obnubilation, the site of headache, the staggering gait not of cerebellar type, the negative rotation tests. The presumption that the growth is in the left frontal lobe would be rendered certain or not by the observation of one of the convulsions, and perhaps by a study of the nature of the amnesia.

The exploratory operation gives the certainty of great relief and the possibility of extirpation of this growth and cure of the patient. This was arranged for, but before it could be done the patient's

husband wrote that she was under other care, and that he did not desire it; and she has passed from observation.

Case III.—A woman, aged fifty-five years. The family physician relates that two or three years ago he diagnosticated "petit mal." During the attacks a cold sensation mounted the limbs of the left side and she fainted. She had dizzy attacks, headaches, false reminiscences, left hemiparesis, and weakness of memory. A year ago a consultant took charge of the case. He informs me that the unconscious attack lasted only a few seconds, and was not followed by dulness; that the left-sided paresis became worse and better by turns; that expulsive vomiting had been recurring for some time; that she was disorientated and confused, and that the pulse rate varied markedly. No diagnosis had been made.

The relation of these signs itself points most strongly to intracranial neoplasm, and an exploratory operation would have been justifiable upon them alone.

Sensibility. Upon examination I found an impairment of the attitude sense on the left arm and enlargement of the circles of Weber on both hands, forehead, and tongue. Hypesthesia to cold on left lower leg and toes. No marked impairment of vision or restriction nor inversion of fields, and no edema of the optic papillæ. Sometimes she confessed to smelling a hop-like odor in the mornings. The deep reflexes were all exaggerated; the abdominal reflexes were absent, and the left plantar was incomplete.

Motility. The motility was in general feeble, and her mental state prevented quantitative reliable tests. There was distinct facial asymmetry, the left side being weak. There was no strabismus, no nystagmus, nor inequality of pupils. The diadokokinesis was impaired, especially on the left side. The contralateral synergic response was diminished in the left lower limb. The attacks occur after a headache and a creeping numb feeling in the back of the neck, the arm, and leg, which lasts for a few days. No dyspraxia could be elicited.

Intelligence. Mental hebetude was distinct. For example, she called a quarter a nickel, although she disproved asteriognosis by declaring that she felt the milling on the edge. She was much disorientated, not even knowing the date or recognizing people she well knew.

The findings in this case leave no doubt of some slow invasion of the right frontal lobe. Its nature can only be determined by exploration. An operation for the purpose would at the same time relieve the patient, even though an irremovable growth were revealed. I so advised, but it has not been done on account of the timorousness of the physicians in charge of the case, whose failure to make a diagnosis for two years may be responsible for their own opposition to an operation now.

I could cite several other cases, but do not wish to prolong an

argument which should already be so clear as to admit of no misunderstanding, and the conclusion of which I may restate as the imperative necessity of an early neurological examination of any patient complaining of vague intracranial discomfort, symptoms loosely called neurasthenic or hysterical; progressive weakness; clumsiness or dulling of sensibility in any part of the body; nausea and dizziness; dimness of vision not due to refractive errors, especially if evanescent; peculiar subjective sensations of taste and smell; mental dulness; loss of memory or change of disposition. It is only by this means that the surgeon will gain access at a period early enough for extirpation of an intracranial neoplasm.

REFERENCES.

Williams, Intracranial Neoplasms, Remarks on Diagnosis, Lancet-Clinic, April 30, 1910; Charlotte Med. Jour., July, 1910.

Diagnosis of Disorders of Cerebellar Apparatus, Arch. f. Diag., N. Y., January, 1910.

REFLECTIONS ON SOME CRITICISMS OF THE PSYCHOANALYTIC METHOD OF TREATMENT.[1]

By Ernest Jones, M.D. (Lond.), M.R.C.P. (Lond.),

Associate in Psychiatry, University of Toronto; Director of the Ontario Clinic for Nervous and Mental Diseases.

Whatever else may be said about Freud's psychological theories and the psychoanalytic method of treatment none has denied the great significance of them in modern thought. It is becoming increasingly difficult for anyone interested in the problems of psychopathology, or, indeed, of psychology in general, to remain aloof from these theories, or to avoid ranging himself either in support of or in opposition to them. It would be out of place here even to attempt to indicate the significance they have for the sciences of sociology, mythology, and anthropology, as well as for psychology proper, but I wish at the outset to point out how necessarily fallacious it must be for anyone to reach any dogmatic conclusion as to their value so long as he confines himself to only one of their aspects. One of the best tests of the value of any new theory is to find out how fruitfully it can be applied, and the extent of the regions over which Freud's principles have been found to be valid—in regions relating to the activities of past ages as well as the present, to the psychology of the normal, of the neurotic, and the insane—is, perhaps, the most definite piece of confirmatory

[1] An address delivered before a joint meeting of the Chicago Neurological Society and the Chicago Medical Society, January 18, 1911.

evidence of the truth of them. In this paper, however, we are concerned with only one aspect of Freud's work, namely, the method of psychoanalysis that he has devised for the treatment of the psychoneuroses.

Those who hear or read for the first time of the psychoanalytic method may broadly be divided into two classes—namely, those whose interest is aroused though their intellect may be puzzled, and those who from the outset are repelled and adopt a hostile attitude. The line between the two classes is not a very sharp one, for I think it is true that no one has ultimately adopted the theory and practice of psychoanalysis without first having to overcome various difficulties and obstacles in his own mind. One might, therefore, more fairly say that readers show a great variation in the extent to which, in the further study of the subject, they are prepared to waive their own inevitable prejudices. To some the conclusions reached by Freud seem so grotesquely unlikely, and the objections to his method so obvious, that they do not feel it worth while to pursue the subject any farther. Others, equally conscious of the objections that immediately arise in their mind, are sufficiently openminded or tolerant to think it possible that perhaps their difficulties are due to their insufficient grasp of the matter, and that adequate explanations may exist which they will find if they pursue their inquiry. This latter position has this *a priori* consideration in its favor—namely, that many of the objections commonly raised are so extremely simple and obvious in character that it would be a highly remarkable occurrence if a man of Freud's recognized intellectual power had overlooked or ignored them. This consideration becomes the more probable when one recollects that assent has been given to the chief of Freud's conclusions by such men of scientific eminence and sane judgment as Professors Bleuler, August Hoch, Jung, Adolph Meyer, and Putnam. In view of this consideration it would seem unreasonable as well as unscientific to refuse to investigate Freud's conclusions, however strange some of them may at first sight appear. We have further to remember that most of us have a much more limited capacity than we flatter ourselves to have of assimilating new thought. As Nietzche well put it, "Mankind has a bad ear for new music." The history of new movements in culture and science is replete with instances that illustrate this sad fact, and yet how rarely does anyone draw the obvious lesson that they themselves will not find it easy to avoid the same danger when the opportunity is offered to them. There are names that live in history only because of the notoriety the bearers of them won by absurd denunciations of new thinkers, and one cannot help thinking that there are men at the present time who will be remembered by posterity for nothing else than their bitter hostility to the new teachings of psychoanalysis.

Of the principles of the psychoanalytic treatment itself I need

here say but little, for I have already on several occasions outlined them,[2] and I may assume that they are fairly familiar to you. Very briefly put they are as follows: It is a principle accepted by all psychopathologists, and no longer in discussion, that psychoneurotic symptoms are the product of underlying, dissociated mental processes, which are unknown to the patient, or, in Freud's sense, unconscious.[3] The symptoms are, therefore, substitutions of unconscious mental processes, and cease to exist when these are made fully conscious. Freud finds that the unconscious processes in question constitute an elaborate network of memories that centre around certain definite dynamic trends, or wishes, which are always of a sexual nature and are always connected with infantile experiences. They are incompatible, or out of harmony, with the rest of the patient's thoughts, and Freud holds that it is the conflict resulting from this fact that is the cause of their being split off, dissociated, or "repressed." They are under the ban of various inhibitions, or "resistances," and the patient automatically strives against acknowledging their very existence. Psychoanalysis is a method, having its special technique, that was devised for the purpose of overcoming these resistances, in order that the unconscious mental processes may be led back into consciousness. The chief aim of the treatment is to give the patient a better understanding of the innermost part of his mind, so that he may be in a position to divert the mental energy that was finding an outlet in the form of neurotic symptoms into more suitable and useful social channels. He cannot do this so long as the energy is locked up by unconscious fixations, so that it is necessary first to free it before it can be applied in healthier directions; it is a question of *reculer pour mieux sauter*.

The objections that have been brought against this method of treatment may for present purposes be divided into those concerning the practical using of the method and those concerning the theory of it. Most of them can be summarized in the statement that the theory is one-sided and largely untrue, while the application is harmful, impracticable, and unreliable.

It is easily demonstrable that some of the individual objections are, so to speak, not psychologically honest, in that they are only pretexts seized for the purpose of covering deeper ones, and often those who bring the objections are not conscious of the deeper roots of their antipathy to the method. It is, therefore, indispensable that we first say a few words about the relations of the subject to the normal. Freud maintains that the conflicts that lie at the base of neurotic conditions play an important and unavoidable part in the development of every one of us, and that on the out-

[2] Psychoanalysis in Psychotherapy, Jour. of Abnormal Psychol., iv, 140; The Psychoanalytic Method of Treatment, Jour. of Nerv. and Ment. Dis., May, 1910, p. 285.

[3] For an explanation of this term see Psychol. Bull., 1910, p. 111.

come of these conflicts depends whether a given person will be healthy or neurotic. In other words, the difference between a neurotic and a healthy person is not that the former has been subjected to conflict and the latter not, but that the two have reacted differently to similar conflicts. The whole matter is one essentially of childhood. We all know that an infant comes into the world destitute of the various social conventions and inhibitions that play such a large part in adult life. He is originally egoistic; cannot appreciate why his different privileges and impulses have to be regulated or interfered with; has at first no sense of the rights or feelings of others; will attempt to satisfy his bodily needs where and when he thinks fit; craves for food, attention, and other wants, quite regardless of external considerations. His early education consists almost entirely in learning to subordinate his personal desires and interests to considerations that at first only remotely appeal to him—in other words, it consists in constant adjusting of conflicts between internal and external situations. It is only gradually that such barriers are built up as modesty, shame, disgust, horror, moral feelings, and so on. Freud holds that the personal impulses, largely originating in various physical sensations, that are in this way renounced, are far more significant to the child than is generally supposed, and that the compromise thus demanded in its education is often arrived at only at considerable expense to mental health, and is often an imperfect one. A healthy person emerges from this series of conflicts by managing to replace the primary personal activities and interests by external social ones, and the mental energy of the former constitutes one of the main driving forces for the later acquired ones, being one of the chief bases for the whole later character of the person. The normal process, therefore, is repression plus satisfactory replacement, while what happens in the future neurotic is repression plus unsatisfactory replacement. Satisfactory replacement is termed sublimation, the energy, desires, and interests that were originally personal having been sublimated on to an impersonal, social sphere. In the neuroses, on the other hand, the symptoms are really a compromise between open expression of the original impulses and satisfactory replacement of them, or sublimation. In order to bring about normal sublimation, the impulses have first to be freed from their attachment to the neurotic symptoms.

The immediate bearing of these considerations is the conclusion that, as repression of certain primitive tendencies occurs in both the normal and the neurotic, one has to expect that both will show a resistance, which is merely the obverse of repression, against being made conscious of the repressed mental processes. This is, in fact, what is found. The normal person is shocked, and finds it incredible, when he is told of the great frequency with which in their childhood neurotic patients indulged in sexual phantasies in connection with

their parents. The reason is that the same thing is true of the normal, and he is resenting the information about the neurotic because really he is being told a disagreeable truth about himself. It is the people with secret attractions to various temptations who busy themselves most with removing those temptations from other people; really they are defending themselves under the pretext of defending others, because at heart they fear their own weakness. Similarly those who learn psychoanalysis regularly discover that the aspects of it that have most repelled them concern just the strongest and most deeply buried part of their own nature. In resisting these aspects they of course seize at all sorts of superficial reasons and excuses for thinking them untrue, but these are merely the cover for deeper personal grounds, the existence of which they are often unaware. In approaching the subject of the objections to psychoanalysis it is much more important to deal with this general truth than to argue about the superficial pretexts *seriatim*, and that is the reason why I have tried to emphasize it at the outset.

Taking up first the objections to the theory or principles of the treatment, we may first consider the most important—namely, to the effect that Freud attributes an exaggerated importance to sexual factors in the causation of the neuroses. Well, to begin with, there is a frequent misunderstanding in the minds of those who bring forward this criticism. They seem implicitly, if not explicitly, to take Freud's statements as if it meant that, according to him, the psychoneuroses are due to an unsatisfied desire for sexual intercourse, and naturally they triumphantly point to the obvious fact that a psychoneurosis may co-exist with full opportunities for gratification of this desire. Such critics evidently overlook the fact that the sexual instinct comprises a range of activities very much wider than this specific one. The commonest manifestation of the instinct—namely, love—frequently exists without any trace of desire for sexual intercourse, at all events consciously. Then all sorts of other activities, such as perversions, masturbation, and other autoerotic acts, obviously have to be called sexual, although their relation to sexual intercourse is very remote. There are many people, both men and women, who never have any desire for normal intercourse, but investigation shows that they have some other form of sexual life, often a very complex one. Psychoanalytic and other research has clearly shown that the desire for sexual intercourse is a highly specialized manifestation of the sexual instinct, and one relatively late in developing. It is preceded, and accompanied, by a great number of other sexual activities, some of which lead up to it, while others do not. It has been found that the instinct is a composite construction made up of several separate components. Distinct evidences of these components are to be observed even in early childhood, though the form they then take is very distant in appearance from what we call an adult sexual act.

It is impossible to go farther into this matter here, but those interested in it are recommended to read a translation that has just been published of Freud's *Drei Abhandlungen zur Sexualtheorie*.[4]

It has sometimes been asked why sexual disturbances should play such an essential part in the production of the neuroses as Freud maintains. Logically this question should be put after it has been determined whether they do or not, not, as is usually the case, before. It is entirely a question of facts. All I can say here is that no one who has conscientiously, and free from all prejudice, explored the dark regions of the mind where neurotic symptoms arise has any doubt about the essentially sexual nature of them. Freud says:[5] "Ich kann diesen Satz nur immer wieder von neuem wiederholen, weil ich es niemals anders finde, dass die Sexualität der Schlüssel zum Problem der Psychoneurosen wie der Neurosen überhaupt ist. Wer ihn verschmäht, wird niemals aufzuschliessen imstande sein. Ich warte noch auf die Untersuchungen, welche diesen Satz aufzuheben oder einzuschränken vermögen sollen. Was ich bis jetzt dagegen gehört habe, waren Aeusserungen persönlichen Missfallens oder Unglaubens, denen es genügt, das Wort Charcot's entgegenzuhalten, 'Ca n'empêche pas d'exister.'" These words were written six years ago, and others besides Freud are still waiting. Those who have learned the importance of the sexual factors in the neuroses have also come to realize that this fact is not so surprising as it at first appears, for they have learned that the stream that we call the sexual instinct is much broader and deeper than is commonly supposed. The very fact that the greater part of it flows in underground channels creates an extensive illusion as to its extent, but even consciously we cannot doubt that it plays the most important part of all the instincts. From it is evidently derived the main impetus that gives rise to artistic, literary, and poetic productions, and far more of our daily interests and ambitions than we superficially imagine are in reality sublimations from deeper and ultimately sexual sources. The various childhood conflicts above referred to concern almost entirely root manifestations of the same instinct, and anyone who has closely studied the steps by which infantile activities are transformed into non-sexual interests will be not at all surprised that frequently this delicate process does not proceed in the harmonious way that is necessary for the establishment of normal mental balance.

A critic might now remark: Granted all this, admitted that sexual factors are important, and perhaps the most important, agents in the causation of the neuroses; in view of the fact that most of them are unknown to the patient, why resuscitate them, what useful purpose can be served by delving into these disagreeable

[4] Three Contributions to the Sexual Theory, translated by Dr. A. A. Brill. Published in the Journal of Nervous and Mental Disease Monograph Series, 1910.

[5] Sammlung kleiner Schriften zur Neurosenlehre, 2e Folge, S. 103.

memories? This question involves two distinct matters, both of which, however, will be considered together—namely, the psychological explanation of the efficacy of the treatment, and the desirability or not of probing into a patient's innermost sexual life. The answer to the first of these questions was indicated above, when it was pointed out that the translation of the unconscious mental processes to consciousness necessarily signifies the harmonious fusion of mental elements that were previously in permanent conflict, and that the cessation of these conflicts carries with it the lasting cessation of the symptoms; the keynote here is that conscious knowledge of aberrant tendencies means a better and healthier control of them. As I have in a recent paper[6] fully discussed this matter, as well as the puerile allegation that the results of psychoanalytic treatment are due to suggestion, I will not deal with it further here. The second question is evidently based on the strong medical prejudice against handling sexual problems which is shown in so many ways, in the total exclusion of sexual subjects from the medical curriculum, in the marked disinclination to accept the sexual etiology of various diseases, and so on; the long fight, now settled, over the syphilitic origin of general paralysis was a striking instance of this. On the rare occasions when a sexual factor is mentioned in a medical text-book it is usually under the vague and misleading guise of "sexual excesses." This medical prudery is responsible for much harm, both to the profession who are thereby blinded to an important side of pathology and to the patients that consult them. Yet there is no reason to suppose that medical men outside their work are oblivious to the important part that sexual interests play in daily life. I remember on one occasion when I read a paper expounding the psychosexual origin of certain neuroses the opinion was freely expressed that sexual thoughts could not have such a hold on the minds of neurotic patients as I maintained they did. After the medical discussion an informal smoking concert was held, in which the main entertainment consisted in the relating of sexual and skatalogical anecdotes and recitations. I could not refrain from thinking of Heine's well-known stanza from the Die Heimkehr:

> Blamier mich nicht, mein schönes Kind,
> Und gruss mich nicht unter den Linden;
> Wenn wir nachher zu Hause sind,
> Wird sich schon alles finden.

This very nicely expresses the attitude in question. Only at home, in private, or in club life are such topics to be mentioned, not in scientific discussions, in public, "unter den Linden." Yet it is with this intimate, personal, and human side of neurotic patients that we are necessarily concerned, for it is in these deeper recesses of their nature that conflicts and disharmonies arise, not on indifferent and imper-

[6] The Action of Suggestion in Psychotherapy, Jour. of Abnormal Psychol., v, 217.

sonal topics. Evidently a given physician may from personal distaste refuse to investigate such matters, but he should recognize that by so doing he is allowing his personal prejudices to enter into a region where they do not belong—namely, scientific therapeutics. It is as though surgeons should officially proclaim that gynecological therapeutics is too distasteful for medical men to concern themselves with.

Critics who bring forward this objection usually cover their personal disinclination that lies at the root of it by contending that such a procedure is harmful for the patient. In this contention there is an undeniable modicum of truth. No one would maintain that exploration of a nervous patient's sexual thoughts and fancies contains no potentialities for harm, any more than that exploration of a parturient uterus by a village midwife contains no similar potentialities. But it must be remembered that while an investigation of sexual thoughts is not necessarily harmless, it is also not necessarily harmful. In other words, it is a therapeutic procedure that in this respect resembles most others—namely, its potentiality for harm depends almost entirely on how it is carried out. Are the operations of trephining or laparotomy fraught with no risk whatever, to say nothing of the preliminary administration of chloroform? The universal criterion of the advisability or justifiability of carrying out risky therapeutic measures is, or should be, the question of whether the probable good is greater than the possible harm. In such cases as those just instanced it is generally recognized that the potentiality for harm is much less in some hands than in others, and that by the adoption of suitable precautions this potentiality can often be minimized. Now it is contended for psychoanalysis that the various precautions that form part of its technique make it a definitely safe procedure. I should not like to assert that no harm can ever be done during psychoanalysis; it would, indeed, be strange if such a delicate procedure were safer than any other therapeutic procedure. Still, I will say that, in spite of vague statements to the contrary, no case has come to my knowledge where harm has been done to the patient through this treatment, and no one who is familiar with the facts will deny that at all events far more good than harm is done. It is perhaps not out of place here to say a word of warning concerning those physicians who blindly rush into a psychosexual investigation under the name of psychoanalysis, but without any proper training in the matter. It should be obvious that psychoanalysis, perhaps more than any other therapeutic measure, needs a careful, disciplined study before it can be adequately applied or judged.

Another very prominent objection brought against the psychoanalytic treatment is that it is said to be unreliable. This applies partly to the main theory, but especially to the interpretations of the patient's symptoms, utterances, and other mental material. Into the construction of the theory, as into that of all theories, it

is plain that various preliminary hypotheses have entered; but it is maintained that these are the reverse of fanciful, and are always direct and legitimate inferences from facts objectively gathered. Freud himself says:[7] "Ich setze keinen Stolz darein, die Spekulation vermieden zu haben; das Material für diese Hypothesen ist aber durch die ausgedehnste und mühevollste Beobachtung gewonnen worden." The question of actually proving in detail the various parts of the theory is technically an extremely difficult one, both for psychological reasons connected with the meaning of what constitutes scientific proof, and for extrinsic reasons, some of which will presently be mentioned. It has been said that up to the present not sufficient material has been published to establish either the truth of the theory or the value of the treatment. I sometimes think that those who make this statement are unaware of the extent of the material that has been published by the Freudian school. There exist some twenty-five books on psychoanalysis, five periodicals are entirely devoted to the subject, while the number of papers runs into many hundreds. Nevertheless, I freely admit that, in spite of this, not enough observations have yet been published to prove scientifically every inference and interpretation that has been made. On the other hand, we contend that enough has been published to demonstrate the principles of the method, to show the truth of a number of detailed inferences based on the application of it, and thus to establish the right to ask that the other inferences be not denied by critics who have not personally investigated them. A word must also be said about the actual difficulties of publishing cases, which are much greater even than they appear. It is not merely that absolute general precautions must be taken to conceal the identity of the patient, but minor points in the analysis that show the validity of it have to be suppressed, for it is just those that would betray this identity. Then, again, a given interpretation, which can be put in a sentence, may be based on several hours of detailed observation, most of which, the individual utterances of the patient, the tone, the emotional gestures, etc., are impossible to reproduce, although it is just these that inevitably convince one of the validity of the inference drawn.

There are, however, much deeper reasons why many of the individual psychoanalytic interpretations seem very unlikely and often strained. It is evident that the connections between different thoughts proceed, or are supposed to, along paths quite foreign to our normal, more or less logical, thinking processes, and that is why anyone who reads of them repudiates them as highly improbable. It is often forgotten, however, that they cannot fairly be compared with logical thinking, for they represent the workings of unconscious mental processes which are demonstrably quite different from our conscious ones. If, for instance, the conscious atten-

[7] Sammlung, op. cit., S. 100.

tion is designedly distracted during a word-association experiment,[8] it will be found that the intrinsic or logical reactions are largely replaced by superficial ones, especially by sound connections; in other words, the more automatic and less conscious mental functioning becomes the more does it proceed by means of superficial connections of the kind familiar to psychoanalysts. I cannot now go farther into the psychological questions here involved, but I wish to draw special attention to the following important consideration. There is every reason to believe that in the normal as well as in the neurotic there exists a strong general resistance to what may be called symbolic thinking, that is, to mental processes that proceed by means of analogy, superficial connections, and so on; in fact, our logical tendencies rightly repudiate what seems to us to be an absurd, irrational mental procedure. Yet in children and savages, during intoxication and insanity, in superstitions, folklore, and even in poetry, there is no doubt that metaphor, analogy, and symbolism play a very large part, sometimes even as large as they do in unconscious mental processes. A little study of the sources of dream material is enough to convince anyone of how extraordinary are the superficial connections that are formed in their mind quite unknown to them. It is often further asked what guarantee one has in psychoanalysis that a given interpretation is correct. It would take too much time fully to answer this, and I can only assert that in the psychoanalytic technique special provision is made for objective criteria and tests of the truth of any given interpretation; the whole method is the very reverse of the vague, uncontrolled, and speculative procedure it is sometimes represented to be.

Very little need be said concerning the objections to the practicability of the psychoanalytic method of treatment, for to anyone who has assimilated the preceding considerations these objections will answer themselves. It has been said that the method is of extremely limited applicability on account of the time and, therefore, expense involved, of the number of cases that are intrinsically unsuited for the treatment, of the difficulty of using it with hospital patients, and so on. It is quite easy to see that the motive of these objections is to depreciate the treatment, rather than to criticise it seriously, for if the enormous value of it were generally recognized one cannot doubt that these difficulties would lose much of their weight, and would be to a great extent overcome. They can most shortly be met by comparing for a moment psychoanalysis with the sanatorium treatment for tuberculosis, for when this was first instituted exactly the same objections were raised: it was a mode of treatment applicable only to the rich; it involved a great expense of time and money and the training of a special class of physicians; was suitable for only certain selected cases; had considerable

[8] See Jung Diagnostische Assoziationsstudien, 1906, Band i, S. 104, etc.

potentialities for harm, and so on. But when it became realized that the open-air treatment gave the most effective results; that the long duration of it was something inherent in the circumstances; that there was no adequate alternative to it; that, in fact, to ignore it was tantamount to not doing the best for those afflicted with tuberculosis, then the difficulties were rapidly overcome, even for the poorest hospital patients. No doubt it is more satisfactory when we can achieve rapid cures of constitutional maladies, a fact that partly explains the gratification with which the recent Ehrlich-Hata discovery has been received. Still, it must be remembered that from the very nature of things there are certain modes of treatment which it is dangerous to press forward too rapidly, such as the reëducation of the system by means of tuberculin, antirabic and other vaccines. Psychoanalysis, which is essentially a reëducative treatment that modifies mental trends of many years' standing, is just such a treatment where it is impossible to press. As a matter of fact, it compares very favorably in many respects with the sanatorium treatment of tuberculosis, for it requires less time, no special apparatus, buildings, nursing staff, and so on, and, above all, can be carried out in an ambulatory service when the patient is about his ordinary work; indeed, it is very desirable that the patient should have some employment or other interest unless he is totally incapacitated. As to the limited scope of the treatment, we already find in practice that there is a wide range over which it is conveniently applied, and there is no doubt that with the goodwill of the medical profession this range would be very considerably extended; although many intrinsic difficulties exist, many of the alleged ones are largely fictitious, being invented or exaggerated by those who really oppose the treatment on other grounds.

In these few remarks I am fully aware that I have not enumerated all the objections that have been brought forward against the psychoanalytic treatment, and have not even fully discussed any one of those I have mentioned, but I trust I have succeeded in my aim of illustrating the fact that there do exist answers to all of them which in the opinion of those who have properly investigated the matter are entirely adequate.

APLASTIC ANEMIA, WITH REPORT OF A CASE.

By Ralph C. Larrabee, M.D.,

SECOND ASSISTANT VISITING PHYSICIAN TO THE BOSTON CITY HOSPITAL, BOSTON, MASSACHUSETTS.

The red corpuscles of the blood are subject to constant loss and replacement. The best evidence of their continuous destruction

within the body is the continuous flow of bile pigment, a derivative of hemoglobin. Red cells are also lost by hemorrhage, internal and external, slight and severe. In the female, menstruation periodically removes large numbers. In health, of course, an equally constant formation of new corpuscles in the red marrow maintains the number at the normal level.

It is obvious that a deficiency of red cells, that is, a condition of anemia, might result from excess in the destructive processes or from deficiency in the reconstructive functions. There is still much obscurity as to the relative importance of these two factors in the different anemias, but it is probable that in the majority the chief causative factor is increased destruction. As a result of this primary loss of hemoglobin-bearing cells, and by way of compensation for it, changes take place in the marrow, and new corpuscles are poured into the blood in increased numbers. If the loss of red cells is extreme, the stimulation of the erythropoietic functions may be so great that the spleen and other organs not normally concerned in the production of red corpuscles may assume this role, or, more properly, resume it, since it is one of their normal functions in the embryo. Thus, the degree of anemia depends not only upon the severity of the process which is destroying the red cells, but also upon the efficiency of the compensatory regenerative processes. Schaumann[1] compares this mechanism to the way in which the functional efficiency of a diseased heart depends not only upon the severity of the valvular leak, but also upon the degree of compensatory muscular hypertrophy. This paper is a study of certain anemic conditions, in which the compensatory processes are strikingly deficient. In classifying the anemias on the basis of blood examinations, three types stand out with fair distinctness. In their clinical and anatomical features the cases correspond fairly well with the same grouping.

1. The orthoplastic or benign group includes nearly all the anemias secondary to definite morbid states—infections, neoplasms, parasites, chronic intoxications, as well as chlorosis, anemias from acute or chronic hemorrhage, or from faulty hygiene and nutrition. The hemoglobin is reduced in greater proportion than the reds (low color index). Qualitative changes, as seen in smears, are, except for achromia, usually slight in proportion to the severity of the anemia. The average size of the reds is normal or smaller than normal (low volume index), and if nucleated reds are present, they are mostly normoblasts. The anemia itself is often of little clinical importance. Even though severe, it remains essentially benign, and recovery is to be expected with removal of the cause. The yellow or fatty marrow of the adult is changed partially or completely to red, cellular marrow, but it is always normal red marrow, with great preponderance of normoblasts and neutrophilic myelocytes. The change is quantitative rather than qualitative. Obviously such marrow is capable of producing more blood cells

than before. It has undergone compensatory hyperplasia, in order to make up the loss of cells incident to the causative disease.

2. The metaplastic or pernicious blood picture is seen chiefly in the condition or conditions known as pernicious anemia. The anemia produced by infections with Bothriocephalus latus is also of this type, as a rule, and, exceptionally, syphilis, hepatic cirrhosis, and other diseases commonly producing anemia of the benign type will cause instead a metaplastic change. The blood picture is characterized by high color and volume indices, the increased size of the red cells being perhaps the most important feature. The reds show marked changes in form and color. Megaloblasts are almost always found, and they exceed the normoblasts in number. Clinically, the anemia is a much more serious matter than in the first group. Its course is malignant and usually fatal. At autopsy the normal yellow marrow is found to be universally replaced by red celluluar tissue. The changes are not only more intense than in the benign forms, but they are different in quality, the principal histological peculiarity being the increase in megaloblasts.

The known facts concerning those conditions in which the pernicious picture occurs as an obviously secondary manifestation, particularly in Bothriocephalus infections — conditions which frequently recover when the cause is removed — these facts seem to indicate that the changes in the blood and marrow represent a reaction of the hemopoietic organs to long-continued, perhaps specific, destruction of corpuscles. We do not know whether compensation is more efficient or less efficient than in the orthoplastic cases, so far as the capacity for turning out new cells is concerned. Nor can we say, at least in regard to some, that the marrow has not been injured beyond repair. But it is safe to assume that the destruction of red cells has been extreme, and the reaction on the part of the blood-making organs very intense.

3. The aplastic type of anemia is very rare, but its great theoretical significance makes it worthy of the closest study, for much light will be shed upon the nature of the processes by which the erythropoietic organs repair an anemia by a consideration of the exceptional cases in which those reparative processes fail to occur. Clinically the cases show considerable variation. They are alike, however, in the rapid advance of the anemia and the promptly fatal termination. The case presently to be described is, I believe, the only one in which a remission has occurred after the aplastic features have been clearly recognized. Hemorrhages are present in most cases, varying in severity from a few scattered petechiæ to severe hemorrhagic purpura. Other than this the cases show clinically only the features common to all grave, rapidly advancing anemias.

The blood picture is striking and easily recognized. The anemia advances so rapidly that the reds and hemoglobin are usually very low at the first observation. The color index is usually low. The red corpuscles as seen in smears show but slight changes in form

and coloring, even though their numerical reduction is extreme. Nucleated reds are absent or present only in very small numbers. Remarkable changes occur in the leukocytes, the total number being greatly decreased, this decrease involving only the granular forms. The lymphocytes remain present in their normal numbers, while the polymorphonuclear neutrophiles, eosinophiles, and mast cells are absolutely and relatively diminished. This loss of granulocytes is extreme. Mast cells and eosinophiles are often entirely lacking, and sometimes even the neutrophiles can be found only by searching. Platelets are also greatly decreased. The essential features then so far as the blood is concerned are intense loss of red cells and granular leukocytes—those blood elements, and only those, which have their origin in the bone marrow. It is to the marrow then that one looks for an explanation of the disease.

The macroscopic changes in the marrow are not uniform. Sometimes this tissue is yellow or gelatinous, sometimes red and cellular, sometimes mottled, while in rare instances it is replaced by connective tissue (Assmann[12]). The microscopic findings vary correspondingly, but they are alike in the absence of the cells from which the missing blood elements are derived. In the most typical cases the marrow is yellow, and consists of fat. Erythroblasts, myelocytes, and other cells having to do with the formation of blood corpuscles are few in number or completely lacking. Sometimes smears show only debris without cells. Either the marrow is so extensively diseased that it cannot perform its functions, or it fails, for some unknown reason, to do so in spite of the usual stimulus. This inability of the marrow to respond explains why the cases progress so rapidly and why the characteristic blood picture develops.

In certain cases, though they may not be distinguishable, clinically or hematologically, from those already described, the marrow is red and cellular. Microscopically, however, it is neither normal red marrow nor the megaloblastic tissue of pernicious anemia, for both nucleated red cells and myelocytes are lacking or nearly so. In their place there are found only lymphocytes.

Concerning the significance of this change, there has been considerable controversy. Some consider that the presence of the lymphoid tissue is secondary to the disappearance of the normal marrow structures. Others hold that the lymphoid hyperplasia is primary and that the overgrowth of this foreign tissue has led to the disappearance of the normal marrow elements. It is probable that both views are partially correct, some cases being of the one sort, some of the other.

That primary hyperplasia of lymphoid cells may cause aplasia of the red elements is shown by a case reported by Türk.[2] A typical myeloid leukemia changed to what appeared to be lymphatic leukemia. Shortly before death there was a rapid increase of the anemia, with a decrease of white cells to 940 per c.mm., of which

but 6 per cent. were neutrophiles—a typical aplastic picture, involving both the red and white elements. At autopsy the marrow showed lymphoid hyperplasia with almost complete absence of other normal cells. Here there can be little doubt that the aplasia was the result of the crowding out of the blood-forming cells by the overgrowth of the lymphoid tissue.

On the other hand, there are cases, like that of Hirschfeldt,[3] in which it seems equally certain that the lymphoid change in the marrow was secondary to exhaustion of the normal erythroblastic tissue. Here a woman died after an illness of nine months which followed parturition, resembled pernicious anemia, and was marked by profuse hemorrhages. Again, Blumer's case[13] was typical aplastic anemia, associated with lymphoid hyperplasia of the bone marrow. The latter was partly fatty and partly red, from areas of intense lymphoid hyperplasia. The author points out that, as the lymphoid tissue did not occupy the whole marrow cavity, the aplasia could not be explained by mechanical crowding out of the normal elements by the new tissue.

Most cases of aplastic anemia with lymphoid marrow must, however, remain puzzles for the present. Such cases are those of Wolff[4], Senator,[5] and Michaelis.[6] Senator considers them leukemias or pseudoleukemias. Blumenthal and Morawitz,[7] on the other hand, believe the disappearance of the normal elements to be the primary change. Morawitz and Rehn[8] consider the predominant cells in these cases to be not lymphocytes, cells foreign to the marrow, or at least not specific marrow elements, but very early stages of the normal granular leukocytes, that is, promyelocytes or myeloblasts.

A case seen in consultation with Dr. H. H. Germain perhaps belongs to this class. It appeared at first to be noma, and the patient did well for a week after operation. Then he became much worse, and an examination of the blood showed 70 per cent. of hemoglobin, 2,472,000 red corpuscles, and 5000 leukocytes. Nucleated reds were rare, and abnormalities in shape and coloring were but slight. One and only one neutrophilic leukocyte was found during a long search. There were no eosinophiles or mast cells; practically all the white cells were lymphocytes. Perhaps this was lymphatic leukemia with secondary infection of the mouth, perhaps primary infection of the mouth, sepsis, and exhaustion of the marrow, especially in its leukoblastic functions, with disappearance of the granulocytes and secondary or vicarious lymphocytosis.

Such cases are hard to explain. Some of them ought not to be classed as aplastic anemia at all, since the aplasia involved the leukogenic rather than the erythrogenic structures. These two functions are often more or less dissociated in diseases of the class under discussion, sometimes markedly so, as in a case reported by Türk,[9] where, in a case of penumonia and septic endocarditis, the granular leukocytes all but disappeared, the reds remaining normal.

But the anatomical relationship of the cells having to do with the two functions is so close that one can hardly be much affected without involving the other.

There is ample experimental evidence that lymphoid marrow may occur entirely apart from leukemia or related diseases, that it may result from causes commonly leading to anemia, and that the associated blood picture may give evidence of inadequate regenerative activity on the part of the marrow. For the details, the reader is referred to the papers of Blumenthal and Morawitz,[7] Hirschfeldt,[10] Morawitz and Rehn,[8] and Morris.[11] The anemia was produced in these cases by bleeding or by the injection of toxic substances. The results must be applied to the explanation of human aplastic anemia only with great caution, as the blood pictures in the experimental animals differed in important respects, such as the persistence of a normal proportion of polymorphonuclear neutrophiles, from that of the disease in man. But they at least prove that lymphoid marrow does not always signify leukemia.

In some cases it is probable that the lymphoid change is more apparent than real, the lymphocytes not being themselves markedly increased or decreased, but being merely what is left after other cells, normally predominating, have been removed. It is probable that the condition of the marrow previous to the onset of the aplasia has an influence on the histological conditions found at autopsy. One would hardly expect to find the same thing in a previously healthy adult in whom a severe hemorrhage has failed to call forth the usual regenerative changes in the fatty marrow, as in a child, or in a person in whom aplasia is but a terminal event in an illness of a sort usually associated with red marrow. In fact, several writers refer to the lymphoid tissue in their cases as being loosely packed, and suggesting normal marrow minus everything but lymphocytes, rather than the intense infiltration of leukemia.

Concerning the pathogenesis of aplastic anemia, it is conceivable that such a condition might arise from incapacity of the blood-forming organs alone, but in most cases at least there is doubtless also the factor of increased destruction of red cells. Thus, Aubertin[14] distinguishes "anémie *par* anhématopoiése" and "anémie *avec* anhématopoiése." Anemia *from* anhematopoiesis is seen only in leukemia or other destructive disease of the marrow. Simple primary aplasia of the marrow does not exist, and, except in these conditions, aplastic anemia is always anemia *with* anhematopoiesis, one of the usual causes of anemia always being present. This view is also held by Pappenheim.[15] Hemorrhage, sepsis, the unknown noxus of pernicious anemia, or some other agency causes an acute anemia, and the usual compensatory regenerative changes in the marrow fail to occur, perhaps because the same agency that caused the anemia has also injured the marrow. Aplastic anemia is not, therefore, a disease of itself, but merely a condition which may arise in anemias from various causes.

In discussing the etiology of aplastic anemia then we must consider what forms of anemia are likely to have this unfortunate complication. Hirschfeldt[3] describes a case in which typical pernicious anemia changed, after severe hemorrhages, to aplastic anemia. Lavenson[16] discusses at length the relations between aplastic and pernicious anemia, and concludes that the former is always a variety of the latter, the differences resulting entirely from the absence of regenerative processes in the aplastic cases. I doubt, however, whether we are justified in making such a sweeping statement. Existing knowledge of the nature of pernicious anemia is limited. We recognize the disease only by observing its clinical course and its hematological features. In the presence of aplasia, this course is greatly modified, and the blood picture, depending as it does on the peculiarities of the regenerative processes in the marrow, are wholly different where these processes fail. Such cases as that of Hirschfeldt, just quoted, are very rare. Aplasia very rarely occurs in preëxisting pernicious anemia, and the compensatory processes in the marrow usually persist to the end. The patient with ordinary pernicious anemia dies, not because regeneration of the destroyed blood elements fails, but because compensation, intense as it is, is no longer able to keep pace with the still more intense hemocytolysis. If aplastic anemia is merely pernicious anemia without compensatory regeneration, that failure of regeneration exists from the first in the vast majority of cases, and, I repeat, our knowledge of pernicious anemia is still far too meagre to permit us to affirm or deny its presence in the absence of all its common diagnostic features.

There is, in fact, excellent evidence that aplasia of the erythroblastic tissues may complicate anemias which are not of the pernicious type. Ehrlich's classical case,[17] which Lavenson includes in his list, followed acutely after uterine hemorrhage the result of abortion in a previously healthy young woman. Hirschfeldt[18] mentions a case in which the disease followed acutely after a severe hemorrhage.

The following case, which occurred on the service of Dr. E. B. Young at the Boston City Hospital, is of similar origin: A perfectly healthy girl, aged sixteen years, suddenly began to flow profusely. After two weeks she was brought to the hospital and much blood clot was evacuated from the vagina. A condition resembling purpura hæmorrhagica rapidly developed, with hemorrhages from gums and skin. The heart was slightly enlarged, with systolic and diastolic murmurs, and a pericardial friction rub. The hemoglobin was 35 per cent., the red corpuscles 1,338,000, and the leukocytes 6000. There were 12 per cent. of polymorphonuclear neutrophiles, 86 per cent. of lymphocytes, 1 per cent. of large mononuclears, and 1 per cent. of stimulation cells. Platelets were almost absent from the smears. Nucleated reds were rare, and no other abnormalities

in these corpuscles were noted. She died in a few days. Autopsy was refused.

Cases are reported by De Massary and Weil,[19] Steinhaus and Stordeur,[20] Muir,[21] and Blumenthal,[22] in which hemorrhages played an important part, but the circumstances were such as to make it doubtful whether they were the cause or the effect of the blood disease. But when hemorrhage appears suddenly in a previously healthy individual, as a result of such a well-known and adequate cause as abortion, and where aplastic anemia follows directly and acutely, it seems unnecessary to drag in a hypothetical, preëxisting, pernicious anemia.

Quite recently, Selling[23] has reported several cases of anemia of the aplastic type due to benzol poisoning, and refers to experiments, not yet published in detail, in which the condition was produced in animals by the same agent. Furthermore, true secondary anemia may be complicated by aplasia. Thus, Hertz[29] describes 2 cases in which sepsis seemed to be the important etiological factor. Kranz[23] reports a case of aplasia in anemia from Bothriocephalus latus. Schur and Löwy[24] describe a case after corrosive poisoning. Schwartz's case[25] followed renal abscess in a child, the aplasia involving principally the leukoblasts. One of Blumenthal's[22] had syphilis. Berberis' case[26] followed miscarriage, with hemorrhage and sepsis. Finally, Dickson,[27] in his monograph on the bone marrow, describes and figures degenerative conditions in the marrow of several long-standing cases of sepsis, tuberculosis, and cancer, which he attributes to increased activity followed by exhaustion of the blood-forming cells. Unfortunately, no blood reports are given.

In conclusion then it seems altogether probable that aplasia of the marrow may occur in post-hemorrhagic or destructive anemias of any sort, possibly as a result of direct action of the same toxins which destroy the blood cells, possibly from exhaustion by overactivity.

The prognosis in aplastic anemia is unfavorable. With the possible exception of acute leukemia, no disease of the blood is so rapidly and uniformly fatal. So far as I am aware, the case to be reported is the only one in which even temporary improvement has occurred.

Treatment has not been effectual. It does not seem likely that any drug will have the least influence. Direct transfusion should certainly be done when the condition complicates hemorrhage, and, perhaps, in other cases following acutely after definite causes.

For the privilege of reporting the following case, as well as for material assistance in studying it, I am indebted to Dr. C. B. Wormelle, of Brighton, Massachusetts:

Sherman S., aged five years, had been ill six weeks when I first saw him in consultation with Dr. Wormelle, on June 15, 1907. There was nothing in his past history of importance, except an attack of measles, with pneumonia, several years before. At the beginning of his illness, he was sleeping in the same room with his

brother, who was at the time suffering from chronic suppurative disease of the bones, with discharging sinuses on the face and in the groin. This child has since recovered. Sherman's symptoms were those of a rapidly advancing anemia, with irregular fever, often as high as 103°. His only complaints were weakness and slight abdominal pain. Examination showed a well-developed but poorly nourished boy. He was apparently very ill, stupid, and irritable, resisting examination. He was very pale, with the yellow tinge so suggestive of pernicious anemia. There was a small hemorrhagic spot on the forehead, which his mother could not explain. The heart was slightly enlarged to the left, and presented systolic and diastolic murmurs. The other viscera were normal. There was no enlargement of the liver, spleen, or lymph nodes. There was no edema. The reflexes were normal. There was a slight Kernig sign, but no rigidity of the neck. The eardrums were normal.

On the basis of the blood examination, a diagnosis of aplastic anemia was made, and an unfavorable prognosis given. The unexpected happened, however, and he promptly recovered. His color became good, the heart murmurs disappeared, and at the end of a month he was apparently as well as ever.

In October of the same year a large ecchymosis suddenly appeared in the right groin, and the old symptoms recurred, with rapidly advancing anemia and high fever. When seen by the writer, on November 11, his condition was about the same as in June. The spleen was still normal, but the liver edge was palpable, and lymph nodes, varying from a quarter to a half inch in diameter, could be felt in the groins, axillæ, and neck. A slight nose-bleed had just occurred. Minute punctures made for the purpose of obtaining blood either bled profusely or caused tender, hemorrhagic swellings. On November 16, about a month from the beginning of the relapse, the child vomited a large amount of blood, and died.

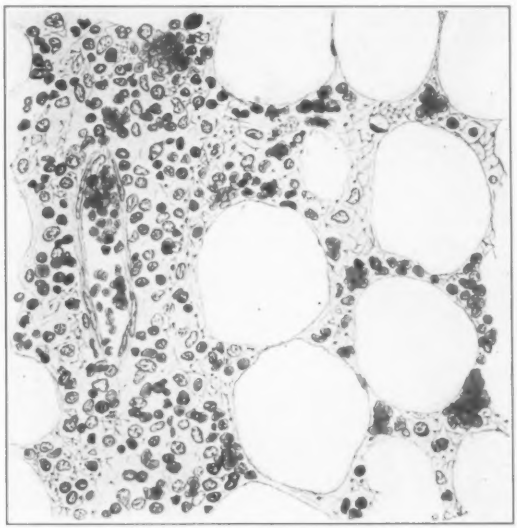
	June 10.	June 15.	July.	November 11.
Hemoglobin, per cent.	40% (Tallqvist).	12% (Sahli).	85% (Tallqvist).	25% (Sahli).
Red corpuscles	2,000,000	790,000	3,700,000	1,500,000
White corpuscles	6000	1970	Decreased	5030
Polynuclear neutrophiles	....	8.8 per cent.	....	3.2 per cent.
Lymphocytes	....	88.0 "	....	90.8 "
Large mononuclears and transitionals	....	1.2 "	....	0.4 "
Eosinophiles	....	0.0 "	....	0.0 "
Mast cells	....	0.0 "	....	0.0 "
Myelocytes (neutrophiles)	....	0.4 "	....	0.4 "
Stimulation cells	....	1.6 "	....	5.2 "
Erythroblasts per c.mm.	....	24	....	
Megaloblasts seen	0	2	0	0
Normoblasts seen	0	1	0	1
Changes in reds	....	Slight	....	Slight
Size of reds	....	Small	....	Vol. index 0.69
Platelets	....	Much decreased	....	Almost absent

The blood findings are shown in the table. The first two columns show the rapid advance of the anemia, the reds falling from 2,000,000 to 790,000 in five days. In spite of this low figure, the reds appeared almost normal in smears, and nucleated forms were present only in small numbers. The decrease of platelets was obvious. Leukopenia was striking, and involved the granular leukocytes to a marked extent, the actual number of polymorphonuclear neutrophiles per cubic millimeter being but 173 instead of the normal 4000 to 6000. Eosinophiles and mast cells, likewise formed in the marrow, were absent. The third column shows the extent to which the blood recovered in the remission. The final column shows the blood in the relapse, a week before death. The anemia had not yet become remarkably severe. The volume index, which expresses the average size of the red corpuscle referred to 1 as normal, was estimated after the method of Capps,[30] except that the corpuscles were sedimented spontaneously instead of by the centrifuge.[32] The low figure is like that of benign anemia, and in contrast to the high values commonly seen in pernicious anemia. The leukocytes are higher than before, but there is the same decrease of the granular forms, the absolute number of neutrophiles being 161 per c.mm. The lymphocytes (4577 per c.mm.) are not beyond the normal limits for a child aged five years. On the whole, these are typical pictures of aplastic anemia.

An autopsy was performed by Dr. Timothy Leary, who, it is much to be hoped, will later report upon it at length. There were no important abnormalities except those having to do with the blood and blood-forming organs. The esophagus contained a large clot, and there were small petechial spots on the mucous and serous surfaces. The marrow of the vertebræ was pale red. That of the femur was yellow and jelly-like, and mottled with red. The microscope showed typical fat tissue with abundant hemorrhage between the fat cells, apparently the cause of the red mottling seen in the gross specimens. Here and there about the arterioles there were small islands of nucleated cells. These were almost exclusively lymphocytes. Nucleated red corpuscles, normally so abundant, were very rare, and were small in size. A single group of megaloblasts was found after long search. Granular leukocytes were also rare. Neutrophilic myelocytes, so abundant in normal red marrow, were only found after careful searching. But one eosinophile was seen. There was a diffuse infiltration of the lymph nodes and of the interlobular connective tissue of the liver, with lymphoid cells somewhat suggestive of lymphatic leukemia. The spleen, otherwise normal, contained immense numbers of large epithelioid cells, within which were masses of yellowish-brown pigment.

The red color of the marrow was not due to the presence of regenerative cellular changes, but to hemorrhages, which were present in many other parts of the body as well. The marrow was essen-

tially aplastic, the aplasia involving the leukoblastic (granulocytic) as well as the erythroblastic elements. There is, to be sure, a *relative* increase of lymphocytes in the marrow as well as in the other tissues, somewhat suggestive of lymphatic leukemia, but it must be remembered that the patient was a child, aged five years. At this age the lymphatic apparatus is very active and readily responds to stimuli, and the number of cells of this type is, both in the blood and in the tissues, greater than in the adult. No diagnostic significance then can be given to this finding, nor to the slight increase of lymphocytes in the last examination of the blood. The case is surely not one of replacement aplasia. The amount of lymphoid tissue is at no point great enough to have



Bone marrow of Sherman S. Aplastic anemia. Cell island about a small artery on left of picture. Cells mostly lymphocytes. Hemorrhage into tissues between fat cells.

influenced by its presence other normal structures. The microscopic picture is red marrow minus everything except lymphocytes. Disregarding the hemorrhages, the prominent feature is replacement of normal cells by fat. The lymphocytes have not been thus replaced, but they are themselves little if at all increased.

The rarity of giant cells and the concomitant rarity of platelets is in complete accord with the views of J. H. Wright as to the origin of these bodies.[31]

It is difficult, perhaps at present impossible, to determine the primary cause of the anemia in this case. The yellow color of the skin, the marked remission, the iron-containing pigment in the

spleen, and the single group of megaloblasts found in the marrow suggest pernicious anemia, but, considering the uncertainties and qualifications imposed by our present lack of knowledge of the disease or diseases known by that name, it is perhaps safer not to venture a diagnosis. Some unknown agency caused this child to become anemic, probably through destruction of red corpuscles. For a time the marrow failed to react, perhaps because it suffered injury from the same noxus. The remission was probably due to temporary recovery on the part of the marrow, since it seems unlikely that this tissue in the condition found at autopsy could have replaced the lost cells so promptly, even if the hemolytic agent ceased its activity. Finally, aplasia again supervened, with or without exacerbation of the destructive process, and from this second attack the child died.

REFERENCES.

1. Schaumann. Sammlung klinische Vorträge, 1900, No. 287.
2. Türk. Verhandlungen des Kongresses f. innere Medizin, 1906, xxiii, 589.
3. Hirschfeldt. Berliner klinische Wochenschrift, 1906, xliii, 545.
4. Wolff. Berliner klinische Wochenschrift, 1905, xlii, 36.
5. Senator. Zeitschrift f. klinische Medicin, 1904, liv, 1.
6. Michaelis. Zeitschrift f. klinische Medicin, 1902, xlv, 87.
7. Blumenthal und Morawitz. Deutsches Archiv f. klinische Medizin, 1908, xcii, 25.
8. Morawitz und Rehn. Deutsches Archiv f. klinische Medizin, 1907–08, xcii, 109.
9. Türk. Wiener klinische Wochenschrift, 1907, xx, 157.
10. Hirschfeldt. Deutsches Archiv f. klinische Medizin, 1908, xcii, 482.
11. Morris. Johns Hopkins Hospital Bulletin, 1907, xviii, 200.
12. Assmann. Beiträge zur pathol. Anatomie und zur allgemeine Pathologie, 1907, xli, 565.
13. Blumer. Johns Hopkins Hospital Bulletin, 1905, xvi, 127.
14. Aubertin. Semaine médicale, 1908, xxviii, 337.
15. Pappenheim. Folia Hematologica, 1908, vi, 104.
16. Lavenson. Amer. Jour. Med. Sci., 1907, cxxxiii, 100.
17. Ehrlich. Charité Annalen, 1888, xiii, 300.
18. Hirschfeldt. Fortschritte der Deutschen Klinik, 1910, i. 298.
19. De Massary et Weil. Société médicale des hôpitaux de Paris, 1908, xxv, 872.
20. Steinhaus et Stordeur. Arch. de méd. expériment. et d'anat. pathologique, 1908, xx, 805.
21. Muir. British Medical Journal, 1900, ii, 909.
22. Blumenthal. Deutsches Archiv f. klinische Medizin, 1907, xc, 132.
23. Kranz. Folia Hematologica, Supplement, 1907, iv, 245.
24. Schur und Löwy. Zeitschrift f. klinische Medicin, 1900, xl, 412.
25. Schwartz. Wiener Gesellschaft f. innere Medizin, 1904, June 23. Quoted by Türk.
26. Berberis. Rif. Med., 1908, xxiv, 849. Abs. in Folia Hematologica, 1910, ix-i, 206.
27. Dickson. The Bone Marrow: A Cytological Study, London, 1908.
28. Selling. Johns Hopkins Hospital Bulletin, 1910, xxi, 32.
29. Hertz. Wiener klinische Wochenschrift, 1908, xxi, 1363.
30. Capps. Journal of Medical Research, 1903, x, 367.
31. Wright. Boston Med. and Surg. Jour., 1906, clvi, 643; Jour. of Morphology, 1910, xxi, No. 2.
32. Larrabee. Journal of Medical Research, 1911, xxiv, 15.

GRANULOMA PYOGENICUM (BOTRYOMYCOSIS HOMINIS OF THE FRENCH).

By Richard L. Sutton, M.D.,

ASSOCIATE PROFESSOR OF DERMATOLOGY AND CHIEF OF CLINIC, UNIVERSITY MEDICAL COLLEGE; DERMATOLOGIST TO THE GERMAN HOSPITAL AND TO THE SWEDISH HOSPITAL, KANSAS CITY, MISSOURI.

In 1897 Poncet and Dor[1] first described a neoplastic, fungating, pedunculated growth occurring in man, in some respects similar to the so-called botryomycotic tumors at times seen in domestic animals. The growths were pea- to nut-sized, reddish in color, and developed by means of a pedicle from the derma. Histologically, their structure was that of a typical granuloma.

Poncet and Dor found numerous yellowish, mulberry-shaped masses of cocci, which they believed to be identical with the botryomycosis (Bollinger). Their findings were partially confirmed by Nocard and by Leclainche, but later investigators, including Bodin,[2] Crocker,[3] and Hartzell,[4] have shown that these bacterial collections consist entirely of ordinary yellow staphylococci. Why the presence of these organisms should occasionally give rise to this particular type of abnormal cell proliferation is not known (the fibro-adenoma theory of Carriere and Potel, which was founded on the accidental occurrence of a coil gland in one of the tumors, is unquestionably erroneous, and has been generally rejected), but the phenomenon is probably similar in many respects to that observed in the highly vascularized tissue which is commonly known as "proud flesh." Owing to the location of the microörganisms, it is practically impossible to bring about a cure by means of the topical use of antiseptics alone, and a lesion may persist for months in spite of treatment. The pedicle is composed of connective tissue, with numerous bloodvessels, and profuse hemorrhage results when it is severed. Even when the growth is removed in this manner it promptly recurs unless the base is thoroughly cauterized.

The condition is not an extremely infrequent one, and the following typical instance is reported, not because of its rarity, but more for the purpose of calling attention to the clinical features of the lesion, in order that it may be more readily recognized when encountered.

H. M., aged nine years, female, schoolgirl. Referred to me by Dr. J. M. Allen, of Liberty, Mo.

Family History. The cutaneous history of the family is negative.

[1] Trans. Congrès de Chirurg. de Paris, October, 1897.
[2] Ann. de dermat. et de syph., iii, 289.
[3] Diseases of the Skin, 3d edition, ii, 1081.
[4] Jour. Cutan. Dis., xxii, 520.

Personal History. The patient is a native of Missouri, and a resident of Missouri City, Mo. She has never had a serious illness, and has never before suffered from a skin disease.

Present Illness. Seven weeks prior to the time of consulting me a small furuncle developed on the back of the patient's left wrist. A few days later this was opened and drained by Dr. Allen, and an antiseptic dressing applied. The parents were instructed to renew the gauze compresses twice daily, carefully cleansing the part with an antiseptic solution every time the dressing was removed. One week later the patient returned to Dr. Allen for further advice.

FIG. 1.—Lesion on back of wrist.

On examination it was found that a small, bright red, pedunculated tumor, the size of an ordinary pea, had sprung up through the opening left by the furuncle. The skin surrounding the lesion was reddened and tender, and there was considerable suppuration. The tumor itself was plum-shaped, with a smooth, moist surface, and was attached to the derma by means of a slender pedicle about 0.5 cm. in length. The growth gave rise to no pain, but was tender to the touch. A stronger antiseptic was prescribed, to be used in conjunction with an astringent dusting powder. In spite of careful and conscientious treatment, however, the tumor continued to

increase in size, and a suspicion was aroused that it might be malignant in nature.

The patient was a blonde, with a fair, smooth, soft skin. A careful general examination revealed nothing abnormal, except a slight enlargement, with tenderness, of the epitrochlear and axillary glands on the left side. On the dorsal surface of the left wrist was an irregularly outlined area of reddened, inflamed skin, measuring 4 by 5 cm. Near the centre of this patch was a small, mushroom-like tumor, cherry-red in color, and soft and compressible to the touch. The pedicle was attached below to the derma, and was not adherent to the broken epidermal wall which surrounded it above. Cultures made from the pus, by Dr. Frank J. Hall, of this city, showed a pure growth of Staphylococcus pyogenes aureus.

FIG. 2.—Nodule, actual size.

A diagnosis of granuloma pyogenicum was made, and it was recommended that the tumor be excised and the base thoroughly cauterized. The pedicle was snipped off with scissors, and the stump and adjacent tissues deeply frozen by means of Pusey's[5] solid carbon dioxide snow. As an antiseptic, a 1 per cent. aqueous solution of liquor cresol compositus was prescribed, and moist dressings, to be changed twice daily, ordered. It was not necessary to make a second application of the snow, as the wound healed quickly, and all dressings were dispensed with at the end of a fortnight. The excised lesion was fixed and hardened in alcohol, mounted in paraffin, and a number of sections made. For staining purposes, hematoxylin-eosin and polychrome methylene blue were used, with Unna's orcein for elastic fibers and Gram-Weigert for bacteria. The tumor consisted of a globular mass of connective tissue, so filled with dilated bloodvessels as to resemble an angioma. Near the base the covering was made up of irregular masses of swollen polygonal cells, but the greater part of the surface was so necrosed that the character of the component cells was not recognizable. The vessels were enormously dilated, and no signs of endothelial proliferation were present. The cavities were for the most part filled with blood. The intervascular stroma was infiltrated with leukocytes, and large numbers of mast cells also were present. No

[5] Jour. Amer. Med. Assoc., xliv, 1354.

giant cells were to be found. Throughout the growth were irregularly distributed collections of cocci, the number of organisms in each group varying from six or eight to several hundred. No peculiarity in grouping was to be noted, however, and nothing resembling the bodies described by Bollinger was observed. No glandular structures were to be seen.

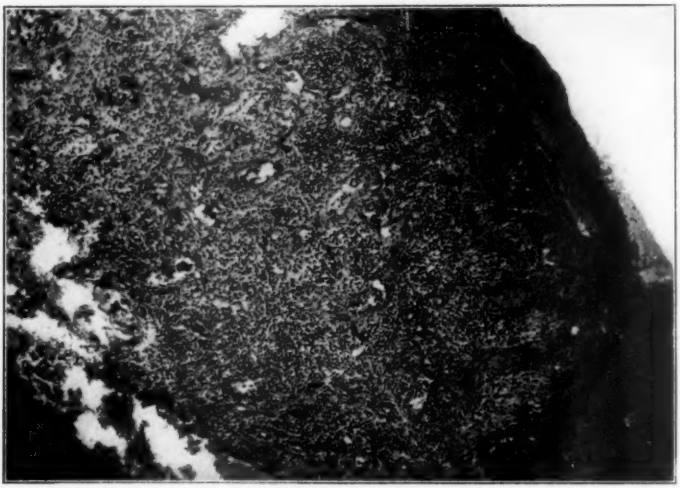


FIG. 3.—Dilated vessels in central portion of tumor, some filled with blood. (Zeiss, obj. C., no ocular.)

ANIMAL EXPERIMENTATION. For inoculation purposes, pus obtained directly from the lesion and staphylococci from the pure cultures on agar were employed. Two healthy adult guinea-pigs were used. A spot on each side of the animal's body was shaved, and the skin thoroughly cleansed with soap and water, followed by alcohol. Superficial lacerations were then made with the point of a knife, and the pus and cultures thoroughly rubbed in by means of a cotton swab on an applicator.

Guinea-pig A. Inoculated from lesion September 6, 1910. Thirty-six hours later the infected area had become red, glazed, and swollen, and on the right side several tiny pustules had developed. On culture it was found that these contained the aureus and a few Staphylococcus epidermidis albus. The left side healed completely within four days, and the area on the right within eight days.

Guinea-pig B. Inoculated from pure agar culture September 12, 1910. The skin was prepared three days previously, in order to secure the presence of an inflammatory condition at the time of

inoculation. Six loopfuls of bacteria and culture material were thoroughly rubbed into the injured integument. Several pustules formed in the course of seventy-two hours, and at the end of five days there was considerable crusting. Nothing that resembled the original lesion developed, however.

It is very probable that granuloma pyogenicum is the result of a staphylococcic infection of the upper corium, and that the involvement of the superficial structures is not directly concerned in its production. The irritation set up by the bacteria and their products gives rise to excessive cell proliferation, and the resulting granulation tissue is forced upward through the opening in the epidermis through which infection of the deeper tissues originally took place.

NOTE: Since this article was written, a valuable and exhaustive communication on the subject has been made by Udo J. Wile.[6]

GENERALIZED ERUPTIONS OF AN UNUSUAL TYPE, CAUSED BY THE ABSORPTION FROM A BELLADONNA PLASTER AND FROM THE OCULAR INSTILLATION OF ATROPINE.[1]

REVIEW OF THE LOCAL AND GENERAL ERUPTIONS CAUSED BY BELLADONNA AND ATROPINE.

BY FRANK CROZER KNOWLES, M.D.,

DERMATOLOGIST TO THE PRESBYTERIAN HOSPITAL; ASSISTANT IN DERMATOLOGY, UNIVERSITY OF PENNSYLVANIA; ASSISTANT DERMATOLOGIST TO THE PHILADELPHIA GENERAL HOSPITAL; DERMATOLOGIST TO THE CHURCH HOME FOR CHILDREN, TO THE BAPTIST ORPHANAGE, TO THE DISPENSARY OF THE CHILDREN'S HOSPITAL; ASSISTANT DERMATOLOGIST TO THE DISPENSARIES OF THE PENNSYLVANIA HOSPITAL AND THE HOWARD HOSPITAL, PHILADELPHIA.

THE subject of belladonna poisoning can, for the convenience of our study, be divided into the local and general eruptions caused by the internal administration of the drug; more or less generalized eruptions caused by the absorption from the local applications of the drug; general symptoms of poisoning, without an eruption, from the local absorption of the drug; local eruptions from the irritation of the local application of the drug; and general eruptions of unusual types from the internal administration or the absorption of the drug from external application.

[6] Unna's Festschrift, Leopold Voss, Hamburg, 1910, p. 333; also Jour. Cutan. Dis., 1910, p. 663.

[1] Read before the Northern Medical Association of Philadelphia, November 24, 1910.

Local and General Eruptions Caused by the Internal Administration of Belladonna. In reviewing the cases of eruption caused by the internal administration of belladonna, one is impressed by the fact that the great majority of the outbreaks are of the erythematous or scarlatinal types. Holthouse reported the case of his son, aged three years and eight months, who took by mistake two drams of a solution of atropine, two grains to the ounce. The skin shortly afterward became hot and dry, and covered with an intensely pruritic rash resembling scarlatina. McNab recorded a case in which a child while out at play ate some berries of belladonna, which was followed shortly afterward by congestion and swelling of the face. In a case reported by Woodman, a woman, aged twenty-one years, took internally, by mistake, a fluidounce of a solution of extract of belladonna rubbed up in water; her face and neck shortly afterward became scarlet. Meredith described a case in which a woman, aged between fifty and sixty years, took, accidentally, methylated belladonna liniment, the quantity being equivalent to twelve grains of camphor and one-half ounce of macerated belladonna root. A few minutes later a scarlet rash appeared on the neck, the upper part of the chest, the forearms, and the hands. Walmsley's patient, a child, aged three years, took four drops of tincture of belladonna; shortly afterward its face became flushed and covered with a scarlatinal blush, interspersed with white spots of irregular size, and shape. Sadler reported a case of idiosyncrasy to the drug. An infant, aged three months, was given a teaspoonful of a mixture containing $\frac{1}{200}$ grain of atropine sulphate to a fluidram; a few minutes afterward the child's face and the upper half of its body became red; this continued for five hours. The same effect was produced with one-half of the dose and with even six drops, the latter being the equivalent, approximately, of $\frac{1}{800}$ grain of atropine sulphate; with the last dose, however, the redness lasted for only one-half hour. Nisbet's patient, a male, aged sixty-one years, took, by mistake, a fluidounce of belladonna liniment, with which was mixed 12 per cent. of chloroform liniment. Three hours later a punctate rash appeared over the chest, the abdomen, and the upper arms. Putnam stated that he had often seen an erythema appear on the face and the chest of babies, lasting about two hours, after even $\frac{1}{32}$ grain of the extract of belladonna had been given. Webber referred to a lady, aged forty years, who had an erythematous eruption on the cheeks after taking $\frac{1}{6}$ grain of extract of belladonna, three times a day, for a few days. Hutinel's patient, a girl, aged twenty-seven months, took fifteen grams of syrup of belladonna; shortly afterward she was attacked by a vivid scarlatinal eruption on the face and the upper part of the trunk. The eruption consisted of large red plaques presenting fine white points. There was very little itching. The eruption lasted for twelve hours, and then disappeared without

leaving a trace. The skin in Lusanna's case became bright red one-quarter of an hour after the administration of atropine, and persisted for one-half hour. The eruption was noted on the face, chiefly in the neighborhood of the ears, and on the hands. Jolly recorded a case in which a mistake was made in a purgative mixture, 44 grains of belladonna being placed in the preparation instead of that quantity of jalap. One hour after taking the mixture redness of the eyes and the face developed, which spread over the entire cutaneous surface, resembling markedly scarlatina. Berenguier reported a scarlatinal eruption on the face and the upper part of the trunk, in a girl, aged twenty-seven months; fifteen drops of the syrup of belladonna had been taken. In Gray's case, belladonna produced a diffuse scarlet efflorescence studded with innumerable papillæ resembling very closely the rash of scarlatina; the upper extremities and the trunk were attacked. In a case which came under Morrow's observation, a child, aged four years, was given an aperient pill containing $\frac{1}{4}$ grain of extract of belladonna. Two hours later there was an excessive flushing of the face, which shortly afterward extended over the body, giving an appearance resembling a "boiled lobster." The eruption disappeared in twenty-four hours, leaving no trace. Köbner observed a characteristic hyperemia of the face from the use of suppositories in the vagina, containing the extract of belladonna.

Eruptions Caused by Absorption from Application of Belladonna Plasters. Judd prescribed a belladonna plaster, about twenty-six inches square, for a strain of the left side and the back. Three hours later the patient's neck and face were flushed to a deep scarlet. Fleming's patient developed the symptoms of poisoning about twelve hours after the application of a belladonna plaster, five by eight inches in size. The plaster had been applied to an area previously inflamed by a strong liniment. The character of the rash on the trunk was not described. Morgan referred to a gentleman who developed a scarlet rash, general in distribution and accompanied by intense itching. A belladonna plaster, at least one foot square, had previously been applied to his loins for lumbago. Harrison's patient gave the history of having applied a belladonna plaster, which caused such irritation that it had been removed; another belladonna plaster was subsequently placed on the abraded area, causing a short time afterward a generalized eruption of a scarlatinal type. Caesar recorded a case in which a belladonna plaster produced an herpetic eruption from the local irritation of the application. Ten years later the same woman, aged thirty-two years, was ordered another belladonna plaster, the itching and burning keeping her awake all night. The whole left side of the trunk became covered by a vesicular eruption situated on an inflammatory base, which outbreak was limited by the median line of the back

and in the front and below by the crest of the ilium. Two days later a mixture of quinine and fifteen drops of tincture of belladonna was ordered, to be given three times daily; the following day a scarlatinal rash appeared on the face, the chest, and the arms. Quinine was given alone and the rash disappeared. Belladonna was then ingested, uncombined, and the eruption reappeared. In the case reported by Griffiths, a woman, aged twenty-three years, applied belladonna plasters to the breasts to "dry up the milk." After the plasters had been *in situ* for some days a well-marked pustular dermatitis was noted on the areas covered by the medication, and a rash of an urticarial type was observed on the arms, the forearms, the anterior surface of the knee-joints, the lower one-fifth of the thighs, and the upper one-fifth of the legs. There was considerable pruritus. Robert Brown referred to a case in which a belladonna plaster caused an inflammation of the skin, with serous discharge.

Poisoning, without an Eruption, Caused by the Application of Belladonna Plasters. In several reported cases the application of a belladonna plaster has given marked symptoms of poisoning, without the development of an eruption excepting, in certain cases, at the site of the plaster. Cases of this character have been reported by Desrosiers, Sym, T. J. Walker, Howarth, Taylor, Douglas W. Montgomery, Burrall, Tansley, Maddox, Clendinnen, and Jenner. In Pye-Smith's case, the advent of an eruption following the application of a belladonna plaster was not mentioned. Goodwin described a curious case in which a woman applied a belladonna plaster for an abscess of the breast, an opening being made in the plaster for the nipple. Her infant died suddenly after nursing at the breast covered with the belladonna plaster, whether from absorption of the belladonna through the mother's milk or from the plaster directly could not be determined. The mother had mild symptoms of poisoning.

Eruptions Caused by Absorption from the External Applications of Belladonna, Exclusive of Plasters. Le Gendre recorded a case in which a woman applied a mercurial preparation for pain in the mammary region, following the same with cataplasms impregnated with belladonna. There was a tremendous reaction, the skin of both breasts, the sternal region, the epigastrium, and the right axilla exhibiting an erysipelatous surface, in certain places ulcerating, with bleeding and suppuration, also vesicular and bullous formation. The axillary glands were enlarged. Fever and ptyalism developed. Six hours after the application of the pomade of mercury and belladonna the body became covered with an eruption resembling scarlatina. Spencer stated that he had treated a case in which a generalized scarlatiniform eruption followed the application, for twenty-four hours, of glycerinum belladonnæ to

the abdomen of a woman. Robinson reported an instance of unusual susceptibility to the local application of belladonna. A man while crushing the leaves and stems of fresh belladonna, accidentally splashed some of the juice on his face, hands, and arms; twenty-four hours later patches of erythema, vesicles, and pustules were scattered over the face. Two years later the man made up four dozen tablets of atropine; the following day his face was covered by an erythematous, vesicular, and crusted outbreak, resembling the dermatitis caused by fierce sunlight. There was marked pruritus. The application of a belladonna plaster, on another occasion, caused a localized vesicular eruption. J. G. Wilson recorded two instances of generalized scarlet efflorescences from the use of belladonna liniment on the breasts of two women, aged twenty-six and twenty-seven years respectively. The eruption appeared in each case on the fourth day after the liniment was first applied, and lasted for a few days after the medication was discontinued. Bloomfield ordered belladonna ointment for a swollen parotid gland, in a girl, aged twelve years; two hours after the second application of the preparation the face became scarlet. Owens describes a case, in a man, aged seventy-three years; a generalized erythematous eruption developed after six drops of a solution of atropine (2 grains to the ounce) had been instilled into the eyes.

Several other papers have been written on this phase of the subject which I have been unable to consult. Enwald and E. H. Brown have compiled articles on poisoning and the eruptions of the skin caused by the external applications of belladonna. Lyman has heading his article, "Poisoning from Belladonna Plaster." Poor takes up the question of skin idiosyncrasy to atropine. Magne has written on the extraordinary idiosyncrasy to the instillation of solution of atropine and belladonna into the eyes.

Poisoning, without an Eruption, from External Applications of Belladonna, Exclusive of Plasters. Several instances of this character have been reported, with more or less severe symptoms of poisoning. Symptoms are produced in the cases of R. G. Brown and Fraser by the external use of the liniment and a lotion containing belladonna. The derangement from the normal was caused in Cooper's patient by the application to the scrotum of an ointment containing belladonna and mercury.

Local Eruptions Caused by the Local Irritant Effect of Belladonna or Atropine. There will be considered under this heading those cases in which the drug itself acted as an irritant to the skin, the dermatitis being in no way caused by the absorption of the belladonna or atropine into the general system through the skin. Julius Donath related a case in which whenever atropine was instilled into the eye an erysipelatous redness developed on the face and the neck. The cheek, the neck, and the shoulder were

swollen, red, and covered with vesicles forming a continuous patch, indistinguishable from an acute vesicular eczema. In Chisholm's patient a strong solution of atropine caused the lids to become puffed and red, with an erysipelatous blush extending down the cheeks. A 1 per cent. solution of atropine sulphate caused circumscribed injection of the conjunctivæ, with swelling of the eyelids and the contiguous cheeks. Duboisia in this patient brought on temporary insanity. Following an instillation of atropine, in Wallace's case, the skin over the eyelids and upper part of the face was swollen, dark red in color, and of a tense, brawny feel; the eyes were closed by the swelling, and a profuse discharge came from the inner canthus. A local dermatitis had previously been produced in this same patient by the application of a belladonna plaster. Lawson stated that he had seen a dermatitis on the cheeks, from the irritant effect of the overflow of atropine from the eyes. Bryce instilled two to three drops of liquor atropii sulphatis (B. P., 1885) into the left eye of a patient, the overflow of the liquid and the tears causing a dermatitis of the cheek. Previous application of a belladonna plaster and belladonna liniment caused a local dermatitis. Mackintosh described an herpetic eruption developing from the local irritant effect of the drug. Belladonna liniment applied to a painful knee-joint caused the outbreak in the first instance, and a solution of atropine instilled into the eyes developed the exanthem on the cheeks, also swelling of the eyelids, on a second occasion. Fialkowski reported a case in which a solution of atropine used in the eyes produced, on the second day, an erythema and eczema of the eyelids. The atropine being continued for ten days, almost the entire face of the patient became covered by a confluent eczema. Liebreich has observed conjunctivitis, erythema, eczema, and pearly granulations of the conjunctivæ after the long-continued use of solutions of atropine, instilled into the eyes. Burdenell Carter has written that when atropine has been long in use, even the purest and most neutral solution is apt to cause local irritation in some individuals, and this irritation is usually more manifest in the eyelids, especially the lower, and on the adjacent cheek.

Local Idiosyncrasy to Atropine (Author's Case). Case I.—A negress, aged twenty-three years, came to the skin dispensary of the Pennsylvania Hospital in 1907, with an eruption involving the entire left cheek, the left side of the nose, and the left lower eyelid. The eruption consisted of innumerable miliary and larger vesicles on an acutely inflammatory base; there was considerable crusting, the latter being honey-colored. The left eye was closed by the marked swelling. There was considerable amount of itching, The eruption developed after the instillation of a solution of atropine into the eye. In October, 1909, the patient was again seen in the skin dispensary of the University of Pennsylvania Hospital,

with an acute vesicular eruption; almost the exact counterpart of that seen on the first occasion. The same history was again elicited. The eruption in the first instance lasted for six weeks and in the second attack for two weeks (Fig. 1).

FIG. 1.—Case I.

UNUSUAL ERUPTIONS CAUSED BY THE INTERNAL ADMINISTRATION OF BELLADONNA. Tardieu stated that occasionally petechial spots may be caused by the ingestion of belladonna. Boecke recorded an instance in which ¼ grain of extract of belladonna, ordered three times a day for a consumptive patient, after the second dose produced an erythema and partial gangrene of the skin of the scrotum. Crocker referred to a case, seen with Whipham, in which a man of forty years wore a belladonna plaster for a week and then took two 7-drop doses of tincture of belladonna. The next day the hands and the feet were swollen, red, and tense; the palms were deep red, with thickening of the epidermis; the soles were less affected; over the knuckles and all points of pressure the redness was intense, and capillary pulsation could be demonstrated by flexing the joint.

Dreyfous reported a case, of considerable interest, of a man, aged thirty-two years, with paralysis agitans; he was given belladonna in ascending doses; after 15 c.c. had been taken, which was followed by a vapor bath, intense itching developed, keeping the patient awake all night. On the next morning the patient's skin exhibited a somewhat generalized eruption. There was a diffuse vivid redness of the lower part of the face resembling erysipelas.

The eyelids were enormously swollen, and small vesicles were noted on the edges of this swelling. There were small nodules on the lobe of the right ear, and the left ear was swollen and red. The posterior portion of the neck, the external portion of the thighs, and the knees were scarlet. The eruption was of the type of the papular erythema on the extremities. There was a diffuse erythema in the neighborhood of the articulations of the elbows, more of a rose tint, but also slightly on the yellowish color. The skin on the dorsal surface of the forearms was dry, wrinkled, infiltrated, red, and formed of pinkish disseminated points; the surface was crusted from the desiccation and breaking of the vesicles. The skin at the bend of the elbows was moist and encircled by crusts and scales. There was a red sinuous line on the dorsal surface of the wrists. Irregularly shaped pinkish patches were found on the dorsal surface of the hands and the fingers, with intervening areas of sound skin. Small acuminate red points surmounted by a vesicle were noted on the back; in some places the eruption had run together, forming red, erythematous, disseminated large plaques. Patches of a light rose color were seen on the abdomen and the chest; these later plaques consisted of innumerable rose-tinted small points, giving the patch a uniform color. The anus was surrounded by large erythematous areas, with considerable crusting. The prepuce and the scrotum were edematous and very much swollen; the irritation caused frequent erections. No eruption was present on the lower portion of the cheeks, the chin, the nose, the palms of the hands, the shoulders, the legs, or the feet. The mouth was dry; the throat normal. The pulse and temperature were unaffected. The pupils were normal. Itching was intense. The eruption almost entirely cleared up in four days.

Gaucher, Boisseau, and Desmouliere reported an interesting case of idiosyncrasy to two separate drugs—hydrocyanic acid and belladonna. The patient was given for abdominal pains $\frac{7}{100}$ mg. of hydrocyanic acid; twelve hours later a vesicular eruption appeared which extended rapidly over the entire trunk and the upper part of the legs. This lasted for fifteen hours, and then disappeared without desquamation, with no general symptoms. Some time later the patient was ordered a mixture; he took the first dose, containing 5 drops of cherry-laurel water and 4 grams of syrup of belladonna in the evening; during the night a generalized urticarial eruption appeared. The skin was edematous, violet in color, intensely itchy. Desquamation started about midday, becoming generalized; large scales of epidermis fell off, continuing for four days. Ten days later a mixture was given for bronchitis, in which syrup of belladonna was prescribed without the cherry-laurel water; twelve hours later a new pruritic eruption appeared, analagous to the preceding, without consecutive desquamation. Two medicines were, therefore, causal in this case, each having been given sep-

arately, with the same result. The sediment of the urine examined under the microscope showed the presence of numerous crystals of cystine; chemical tests proved the same. The authors consider that this, therefore, was the cause of the patient's idiosyncrasy to the drugs. The presence of cystine crystals showed a derangement of nutrition, an insufficient amount of oxidation, and an abnormal intestinal fermentation. The patient was profoundly shocked by this derangement of nutrition, thus causing the susceptibility to the drugs.

Fallen gives the details of a case in which a somewhat generalized scarlatiniform and eczematous eruption was produced by the ingestion of a combination of opium and belladonna. The eruption was reproduced by giving opium alone. As the belladonna was not administered uncombined, it cannot be positively stated that this drug would have produced any eruption or an outbreak of this type.

Generalized Eruptions, of an Unusual Type, Caused by the Application of a Belladonna Plaster and the Instillation of Atropine (Author's Cases). Case II.—Joseph P., born in Greece, well-built, aged twenty-five years, came to the skin dispensary of the Pennsylvania Hospital March 29, 1909. The patient led an active outdoor life, peddling various commodities in a wagon. He complained that an intensely itchy eruption had appeared three days previously. A generalized eruption was noted on the removal of the clothing, and two large belladonna plasters were found over the scapular region. The history was elicited that eight days before his visit to the dispensary the two plasters had been applied because of a slight cough; five days later the rash appeared; at the time of appearance it was of a bright red color, and very itchy. The patient had had no bowel movement for ten days; he stated that his usual habit was to go but once each week. The skin was dry and hot. The patient complained of having a dry mouth and throat, and being very thirsty. The pupils were only slightly enlarged, reacting somewhat sluggishly to light. The temperature, pulse, and respirations were normal. The face was flushed. Dark red erythematous spots covered almost one-half of the anterior portion of the chest, becoming confluent on the lateral portions. Large dark red areas involved the axillæ; reddish plaques, with a tendency to confluence, involved the anterior and posterior portions of the upper arms and the forearms. Irregularly shaped dark red patches, with intervening sound skin, were found on the dorsal surface of the hands. There was a continuous sheet of eruption extending over the abdomen, the groins, the thighs, and the legs to the knees, of a violet-red color, with a tendency to form areas resembling purpura, but from which most of the color could be pressed (Fig. 2). The upper part of the back exhibited dark red patches, with intervening sound skin, from a dime to half a dollar in size. There was a continuous reddish-violet colored plaque, extending from the lumbar region to

the popliteal spaces (Fig. 3). The lower legs, anteriorly and posteriorly, showed only scattered patches, mostly dime-size or smaller, irregularly round, of a bluish color. At the time of the patient's visit, two days later, the plaster having been removed, the eruption had faded considerably; exfoliation had, however, commenced. Exfoliation was marked on the anterior and the posterior axillary folds, the lower abdomen, the back, the inner surface of the thighs, and the upper legs; it occurred in small flakes and strips. On the

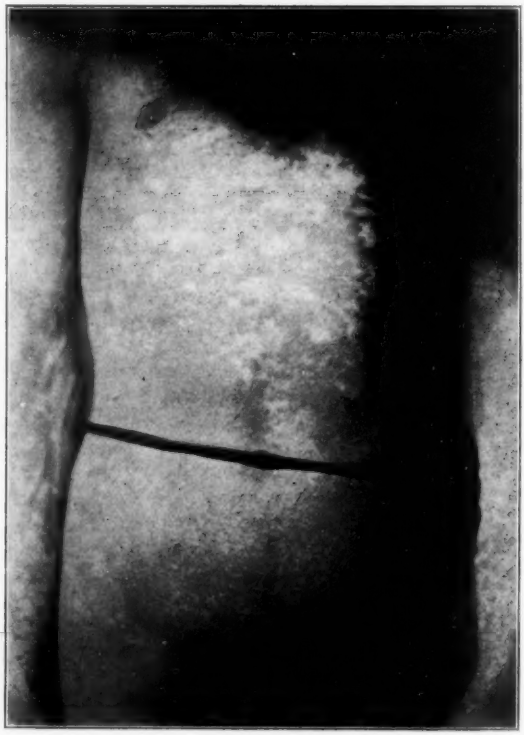


FIG. 2.—Case II. Anterior aspect.

third visit, a few days later, the eruption had almost disappeared, the exfoliation had stopped, but there was some pigmentation where the former outbreak had been.

CASE III.—John O., bricklayer, born in Italy, aged thirty years, came to the skin dispensary of the Pennsylvania Hospital August 18, 1909, complaining of a pruritic eruption of three days' duration. A generalized outbreak was present, consisting of bright to dark

red, erythematous, macular spots, which had become confluent on the chest, the lateral portions of the trunk, the outer surface of the arms, and the thighs. The macular lesions varied from a split pea to a silver dollar in size. The eruption was scanty on the forearms and lower portion of the legs. The hands and the feet were not attacked. The lesions on the lower portion of the trunk, the buttocks, and the thighs were bluish red, of a darker color than those on the upper part of the body. The face exhibited an eruption of a

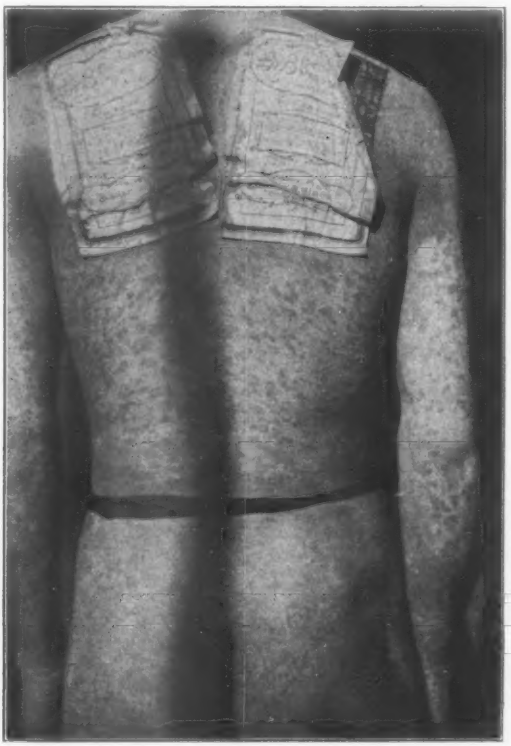


FIG. 3.—Case II. Posterior aspect.

like character, but fainter and of a pinkish color. According to the history, two weeks previous to the appearance of the outbreak, the cornea of the right eye had been injured by a foreign body, just to the right of the pupil, and four drops of atropine had been instilled daily, for a few days. The patient, on his own initiative, had continued this treatment until the appearance of the present eruption. The prescription contained atropine sulphate, 0.15 gram; cocaine

hydrochlorate, 0.3 gram; distilled water, 10 grams. An eye lotion had also been given, containing boric acid, biborate of soda, and water. The pupil of the right eye, into which the solution of atropine had been instilled, was widely dilated. The left eye, into which no drops had been placed, was also somewhat dilated. The throat and mouth, according to the patient, were very dry. The pulse, the temperature, and the respirations were normal. The patient stated that he had been constipated since instilling the atropine.

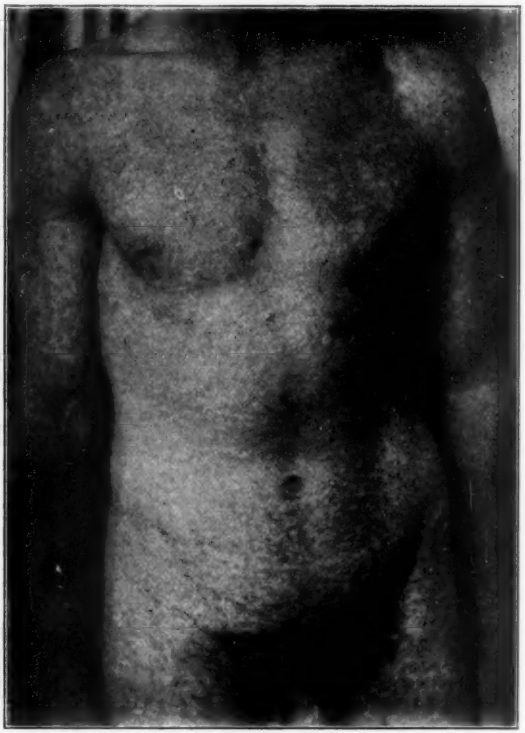


FIG. 4.—Case III. Anterior aspect.

Various symptoms of poisoning were noticed in the great majority of the reported cases, muttering delirium, incoherence in speech, rigidity of the limbs, convulsions, twitching of the muscles, giddiness, staggering gait, delusions in regard to imaginary people and things, cerebral excitement, delirium of laughing and violent types, constant desire to urinate, irritation of the entire urinary tract, rapid pulse, hurried and also deep breathing, hyperesthesia, and hyper-

pyrexia. Mild symptoms of poisoning were noted in the cases of Jenner, Sym, Walmsley, and J. G. Wilson, from the external application of the drug, and consisted of dryness of the mouth, tongue, and throat. Practically no symptoms of general poisoning were noted in the patients of Robinson, Putnam, Webber, Herbert Spencer, and Le Gendre. There was but one fatal case in this series, McNab's patient, a child, aged four years, who died seventeen hours after eating belladonna berries. The temperature reached 110°, a few hours before death.

Although in the great majority of the cases of belladonna poisoning, particularly those of the severe type, the pupils were widely dilated, in some there was only slight dilatation, and in a few there was no mydriasis. In two cases of poisoning, Walmsley's and Sadler's, in which the drug had been administered internally, the pupils were not dilated. Douglas W. Montgomery described a case in which the symptoms of poisoning, from a belladonna plaster, were very severe, but the pupils were absolutely normal. The pupils were also undilated in the cases of Law and Herbert Spencer, the poisoning being caused in the one case by absorption from a belladonna plaster and the other by the local application of belladonna glycerin. The eruption caused by the belladonna plaster in Caesar's patient was unaccompanied by mydriasis. In the cases of poisoning from belladonna plasters reported by Clendinnen and Maddox the pupils were only moderately dilated. In the author's case of belladonna plaster poisoning there was but slight dilatation of the pupils, and the pupil of the eye into which the atropine had not been instilled, in the third case, was only moderately dilated.

The one-sided action of belladonna is a curious phenomenon noted in a few reported cases. Burrall recorded a case in which a woman applied a belladonna plaster upon her right side, causing on the following day mydriasis on the right eye, the pupil of the left eye being absolutely normal. Tansley described two cases of this character: The first patient instilled a solution of atropine sulphate (4 grains to the ounce), once daily, into one ear for tinnitus aurium; twelve days later the pupil of the eye, on the same side as the instillation, became markedly enlarged; the other pupil remained normal. In the second instance, a patient applied a belladonna plaster for pain in the right side, four or five days later the right pupil became enlarged; the left remained undilated. J. Newton Smith mentioned his own case: He took $\frac{1}{2}$ grain of alcoholic extract of belladonna, three times daily; on the second day the right pupil became enlarged; the left was unchanged.

Accidentally a very small quantity of the vomited material got into Holthouse's eye; his son, at that time, was suffering from belladonna poisoning; a short time afterward the pupil became enlarged. The urine and the feces of the child were examined and dilated the

pupils of a white mouse's eyes. The vomited material also caused mydriasis of the eyes of a white mouse.

There is always a hesitancy in describing a unique, or at least an unusual, type of eruption, but the author considers that he is justified in ascribing the two generalized cases to the absorption of belladonna in the one case and of atropine in the other. One could hardly explain the occurrence of two eruptions of almost the same type, excepting that one was much more severe and was followed by exfoliation, on any other theory but that the same drug, or its alkaloid, used in each was causal of the outbreak. There is great regret on the part of the writer that the patients did not return, as directed, so that belladonna could have been administered again to conclusively prove the diagnosis.

CONCLUSIONS. 1. More or less generalized eruptions not only occur from the internal administration of belladonna or its alkaloid, atropine, but also from absorption through the skin of local applications of the same.

2. The skin does not have to be broken or denuded of epidermis for absorption to take place; the symptoms of poisoning, however, develop much sooner if such is the case.

3. The great majority of the cases exhibit an erythematous or scarlatinal type of eruption, which is more frequently found on the face and the upper portion of the body, but in a fair number of cases the outbreak is generalized. Gangrenous, purpuric, and eczematous eruptions have been reported. The author describes a unique, or at least a very unusual, type of outbreak.

4. Although in a great many instances severe symptoms of poisoning are present, the eruption may be unaccompanied by other manifestations of drug absorption.

5. Mydriasis is a quite constant phenomenon; in a few cases, however, the pupils were only slightly enlarged, entirely normal, or unilaterally dilated.

6. The prognosis is favorable; only one death occurred in this large series of cases.

7. Belladonna or atropine may produce a local eruption from the local irritant effect.

I wish to express my thanks to Dr. Charles N. Davis for the privilege of reporting these cases.

LITERATURE.

Holthouse. Medical Times and Gazette, London, 1859, xix, 601.
McNab. British Medical Journal, 1882, ii, 579.
Woodman. Medical Times and Gazette, 1864, ii, 385.
Meredith. British Medical Journal, 1876, ii, 678.
Walmsley. New Orleans Medical and Surgical Journal, 1885-86, n. s., xiii, 979.
Sadler. Medical Times and Gazette, 1868, i, 391.
Nisbet. Australasian Medical Gazette, 1889-90, ix, 114.
Putnam. Boston Medical and Surgical Journal, 1877, p. 142.

Webber. Quoted by Putnam, ibid.
Hutinel. Quoted by Dreyfous, La france médicale, 1877, p. 772.
Lusanna. Quoted by Morrow, Selected Monographs on Dermatology, 1893, p. 437.
Jolly. Quoted by Bérenguier, Thèse de Paris, 1874, p. 34.
Bérenguier. Des eruptions provoquées par l'ingestion des médicaments, Thèse de Paris, 1874, p. 36.
Gray. Quoted by Morrow, Selected Monographs on Dermatology, 1893, p. 438.
Morrow. Selected Monographs on Dermatology, New Sydenham Society, 1893, p. 438.
Köbner. Berliner klinische Wochenschrift, 1877, p. 328 (footnote).
Judd. Journal of the American Medical Association, 1884, ii, 122.
Fleming. Medical Record, 1884, xxv, 68.
Morgan. British Medical Journal, 1866, ii, 620.
Harrison. British Medical Journal, 1872, i, 520.
Caesar. Lancet, 1879, i, 431.
Griffiths. British Medical Journal, 1891, i, 1067.
Robert Brown. Quoted by T. J. Walker, ibid., 1891, ii, 1146.
Desrosiers. L'Union Médicale du Canada, 1886, xv, 13.
Sym. Edinburgh Hospital Reports, 1894, ii, 639.
Walker, T. J. British Medical Journal, 1891, ii, 1146.
Howarth. Lancet, 1894, i, 204.
Taylor. Cincinnati Lancet-Clinic, 1892, n. s., xxviii, 433.
Montgomery, Douglas W. Medical News, 1896, lxviii, 101.
Burrall. Medical Record, 1877, xii, 431.
Tansley. Medical Record, xii, 334.
Maddox. Amer. Jour. Med. Sci., 1893, n. s., cvi, 572.
Clendinnen. Australasian Medical Journal, 1894, n. s., xvi, 446.
Jenner. Medical Times and Gazette, 1856, n. s., xiii, 513.
Pye-Smith. Diseases of the Skin, 1893, p. 180.
Goodwin. Medical and Surgical Reporter, 1871, xxiv, 346.
Le Gendre. Revue pratique d'obstétrique et d'hygiene de l'enfance, 1889, ii, 317.
Spencer. Quoted by T. Colcott Fox, British Journal of Dermatology, 1890, p. 336.
Robinson. Lancet, 1896, ii, 881.
Wilson, J. G. Glasgow Medical Journal, 1871-72, n. s., iv, 198.
Bloomfield. Therapeutic Gazette, 1886, ii, 305.
Owens. Lancet, 1890, ii, 443.
Enwald. Duodecim, Helsingissä, 1889, v, 6 to 9.
Brown, E. H. Medical Record, Calcutta, 1890, i, 243.
Lyman, G. Extr. Rec. Bost. Soc. M. Improve., 1859, ii, 51.
Poor. Budapesti orv. Ujság, 1908, vi, 300.
Magne. Bull. soc. de méd. prat. de Paris, 1883, p. 21.
Brown, R. G. Clinical Lectures and Reports, London Hospital, 1866, iii, 169.
Fraser. Clinical Lectures and Reports, London Hospital, 1866, iii, 171.
Cooper. British Medical Journal, 1877, i, 164.
Donath, Julius. Wiener medicinische Wochenschrift, 1880, xxx, 313.
Chisholm. Maryland Medical Journal, 1881, viii, 375.
Wallace. University Medical Magazine, 1891-92, iv, 298.
Lawson. The Polyclinic, 1904, viii, 39.
Bryce. British Medical Journal, 1898, ii, 987.
Mackintosh. British Medical Journal, 1885, ii, 739.
Fialkowski. Quoted by Morrow, Selected Monographs on Dermatology, 1893, p. 439.
Liebreich. Quoted by Morrow, Selected Monographs on Dermatology, 1893.
Carter, Burdenell. Quoted by T. Colcott Fox, Selected Monographs on Dermatology (footnote).
Boecke. Quoted by Morrow, Selected Monographs on Dermatology, p. 438.
Tardieu. Quoted by Dreyfous, La france médicale, 1877, p. 763.
Crocker. Diseases of the Skin, 1905, 3rd edition, p. 475.
Dreyfous, Ferdinan. La france médicale, 1877, pp. 754, 762, 771.
Gaucher, Boisseau, and Desmouliere. Bull. et mém. Soc. méd. d. hôp. de Paris, 1906, 3e s., xxii, 355.
Fallen. Bulletin société française de dermatologie et de syphilis, 1890, i, 267.
Law. Dublin Medical Press, May 25, 1864, p. 559.
Smith, J. Newton. Medical Record, 1877, xii, 431.

INVESTIGATIONS CONCERNING THE VALUE OF THE MICROSCOPIC EXAMINATION OF THE BLOOD FOR BACTERIA.[1]

By G. A. Fried, M.D.,
ADJUNCT CONSULTING PHYSICIAN, BEDFORD SANATARIUM, BEDFORD HILLS, N. Y.,
AND
A. Sophian, M.D.,
GEORGE BLUMENTHAL, JR., FELLOW IN PATHOLOGY, MT. SINAI HOSPITAL, NEW YORK.

(From the Pathological Laboratory of the Mount Sinai Hospital.)

The following investigation was undertaken at the suggestion of Dr. Libman, to determine, if possible, in what percentage of cases in which blood cultures were positive, bacteria could be found by direct microscopic examination, and also to determine whether organisms were present microscopically in cases in which blood cultures were negative. Altogether 250 cases were studied. The method followed was a combination of those used by previous investigators.[2] We used from 1 to 2 c.c. of blood, which was obtained at the same time that the blood was drawn for cultural purposes, and poured it into 20 c.c. of sterile 1 per cent. aqueous solution of sodium citrate, thus preventing coagulation. The citrate solution as well as all the stains and fluids that were used were passed through a Berkefeld filter in order to remove all contaminating bacteria and granules. The blood was then centrifuged for at least a half hour, and a few drops of the sediment were drawn up into a sterile pipette and deposited on from two to three chemically clean slides. The latter were kept in alcohol until within a few minutes before they were used. The smears were then made and fixed by heat. The blood was then laked with sterile filtered water, after which a 1 per cent. solution of acetic acid was poured over the slides to complete the laking. This was washed off and followed by a routine Gram stain. Various other stains—Giemsa, Unna's alkaline methylene blue, Loeffler's methylene blue, Jennet, fuchsin, and methyl-green-pyronin—were tried, but we found that the Gram stain gave the clearest fields for examination. From one-half to two hours were spent in examining each specimen.

Our method gave better results than that used by Staeubli, viz., collecting the blood directly in 3 per cent. acetic acid, because the fibrin in the sediment interfered with the clearness of the microscopic pictures.

[1] Read before the Section of Internal Medicine, New York Academy of Medicine, November 15, 1910.

[2] Staeubli, Beiträge zum Nachweis v. Parasiten in Blut, Münchener med. Woch., 1908, vol. ii; Rosenberger, The Presence of Tubercle Bacilli in the Circulating Blood, Amer. Jour. Med. Sci., February, 1909.

In cases in which the blood cultures showed the presence of Gram positive cocci, we could, with very few exceptions, and without any knowledge of the result of the blood culture, demonstrate the presence of the organisms within from one to two hours after the blood was withdrawn. By referring to the table, it will be seen that in 97 per cent. of the cases in which the blood cultures were positive, the blood smears were also positive. Frequently the number of bacteria seen on the slide was evidently larger than the number found culturally.

In 12 cases (see appended lists) bacteria were found on the slides although the cultures remained sterile. Of these ,5 were cases of lobar pneumonia, in which one might well expect to find bacteria in the circulating blood. In these smears the cocci which were found appeared as typical lanceolate diplococci. In one case of thrombosis of the lateral sinus the culture remained sterile, but numerous Gram-positive cocci were found on the slides. We might also mention that in 2 cases of acute articular rheumatism organisms resembling streptococci were found in the smears. The only explanation we can offer for negative cultural findings is either inadequacy of methods or devitalization of the organisms due to the bactericidal power of the blood. The results in the cases of rheumatism can be accepted only if confirmed in a large series of cases.

A series of experiments was tried on rabbits in order to obtain comparative results between cultures and spreads. By inoculating rabbits intravenously with varying quantities of cocci we attempted to determine:

1. The maximum length of time after inoculation at which the organisms could still be demonstrated in the blood.

2. The relationship between the number of colonies to 1 c.c. of blood in agar plates, and the number of bacteria found in spreads microscopically.

EXPERIMENT 1. Rabbit. Intravenous injection of 2,000,000,000 Staphylococcus aureus (counted by the Wright method).

After thirty minutes, 8 c.c. of blood aspirated from heart, 4 c.c plated, 2 c.c. in each agar plate, and 4 c.c. collected in sodium citrate solution. Result: Forty colonies to 1 c.c. of blood were counted in the plates after twenty-four hours, and numerous cocci were found easily in the smears.

After twenty-four hours, 5 c.c. of blood were aspirated, 3 c.c. were plated, and 2 c.c. collected in citrate. Result: 1 colony to 1 c.c of blood on the plates, and many cocci found easily in spreads.

After forty-eight hours, 10 c.c. of blood aspirated, 5 c.c. plated, and 5 c.c. collected in citrate. Result: Plates and spreads negative.

After seventy-two hours, animal killed. Cultures from liver and kidney showed Staphylococcus aureus, those from the glands and bone-marrow were negative. The sections showed multiple

abscesses in the kidney with numerous Gram-positive cocci, also a moderate number of cocci in the liver capillaries.

EXPERIMENT 2. Rabbit. Intravenous injection of 1,000,000,000 staphylococci.

After ten minutes, 4 c.c. of blood aspirated from heart, 2 c.c. plated, and 2 c.c. collected in citrate. Result: 350 colonies to 1 c.c. of blood in the plates, and many cocci found easily in spreads. Animal died after aspiration. Postmortem examination negative.

EXPERIMENT 3. Rabbit. Intravenous injection of 2,500,000 streptococci.

After fifteen hours, 5 c.c. of blood aspirated from heart, 3 c.c. of blood plated, 1.5 c.c. in an agar plate, and 1.5 c.c. in a plate of 2 per cent. glucose-agar; 2. c.c. collected in citrate. Result: Plates sterile. A few diplococci found in spreads.

EXPERIMENT 4. Rabbit. Intravenous injection of 35,000,000 staphylococci.

After fifteen minutes, 4 c.c. aspirated from heart, 2 c.c. of blood plated, and 2 c.c. collected in citrate. Result: Eight colonies to 1 c.c. on plates, and cocci found easily in spreads.

From these experiments we conclude that:

1. Bacteria can be found in the spreads when the cultures remain sterile (see Experiment 3).

2. The number of bacteria seen on slides was greater than the number of colonies grown on the plates (see Experiment 1).

3. Many more bacteria circulate in the blood than can be grown culturally, or seen microscopically.

The morphology of the bacteria as seen in the spreads was not always distinctive, but, as a rule, the streptococci were smaller than the staphylococci, and were seen to occur in diplococcus forms, rarely in chains. The staphylococcus appeared singly or in clumps. Identification of the organisms was verified by careful study of the blood cultures.

LIST I.—Cases with Positive Blood Cultures.

(*a*) *Streptococcemia.*

Diagnosis.	No. of cases.	Positive results in spreads
Bacterial endocarditis	5	4
Sinus thrombosis	2	2
Pyarthrosis	5	5
Osteomyelitis	5	5
Erysipelas	1	1
Postabortive general infection	4	4
Cellulitis of hand	1	1
Pneumonia; pericarditis	1	1
Pyopneumothorax	2	2
Total	26	25

(b) *Staphylococcemia.*

Meningitis	1	1
Osteomyelitis	4	4
Infected hemorrhoids	1	1
Pyelonephritis	1	1
Total	7	7

LIST II.—Miscellaneous Cases with Positive Findings in Spreads and Negative Blood Cultures.

Diagnosis.	No. of cases.
Lobar pneumonia	5
Sinus thrombosis	1
Acute articular rheumatism	2
Postpartum general infection	2
Epidural abscess	1
Liver abscess	1
Total	12

LIST III.—Cases with Negative Blood Cultures and Negative Microscopic Findings.

Diagnosis.	No. of cases.
Local infections	30
Appendicitis	8
Mastoiditis	8
Sinus thrombosis (?) mastoiditis	2
Otitis media	1
Puerperal infection	18
Peritonitis	2
Liver abscess	5
Cholecystitis	3
Pyelitis	1
Erysipelas	1
Chronic endocarditis	25
Brill's disease	5
Influenza (unreliable on account of Gram-negative rods)	3
Acute articular rheumatism	4
Gonorrheal arthritis	2
Leukemia	2
Total	120

Besides the cases already discussed, we studied 53 cases of typhoid fever. The method which we employed was found to be useless for this purpose, as Gram negative rods were found not only in the blood of cases of typhoid fever, but also in a large majority of spreads made from the blood of culturally positive streptococcic and staphylococcic cases, and also from the spreads made from many of the cases in which there was no bacteriemia. We can offer no explanation for the presence of these rods, unless possibly that they are hemokonia. We therefore tried to find typhoid bacilli with the ultramicroscope by examining a drop of the sedimented blood, pressing the same between a coverglass and a slide. The method proved to be impracticable on account of the difficulty of distinguishing the bacteria from hemokonia.

In some cases we poured the greater part of the sediment into a tube of bouillon, and placed it in the thermostat for from five to seven hours. The bouillon culture was then centrifuged, and slides were stained for microscopic examination. The number of Gram-negative bacilli in these cases, which were positive culturally, was so large that one could not mistake them for the Gram negative rods seen in small numbers in cases that were not instances of typhoid fever.

In this connection it is evident that because of the presence of Gram-negative rods the method used by us would be impracticable in examining the blood of any case in which the presence of Gram-negative bacilli was suspected. This would, of course, include cases of influenza, and, in view of our findings, we are unable to accept the results published by Canon.

CONCLUSIONS. The studies which we have here outlined will be continued in the laboratory to see if further data can be obtained. At the present time we can draw the following conclusions:

1. Bacteria can be found microscopically in the blood of the large majority (97 per cent.) of cases in which their presence has been demonstrated by blood culture.

2. In a number of the cases (12 in 132, about 9 per cent.)[3] bacteria were demonstrated in the blood which had been found sterile by means of blood cultures taken according to the methods now in vogue in our laboratory. Further studies must be made to determine whether there is any error in our findings.

3. It is probable that in cases of sinus thrombosis and in cases of osteomyelitis, microscopic examination of the blood may be of service in coming to a more rapid conclusion as to the presence of a bacteriemia.

4. Unless all possible precautions are followed in regard to asepsis, and unless all fluids and stains are filtered, the method is unreliable.

5. Microscopic examination of the blood with all the methods that have so far been used is not of service in cases in which the presence of Gram-negative organisms is suspected.

6. Microscopic examination of sedimented bouillon cultures may be of value in making an earlier diagnosis in some cases of typhoid fever.

[3] This includes all the cases with negative blood cultures except the cases of typhoid fever.

DIGESTION IN FEVER.[1]

By John Benjamin Nichols, M.D.,

LECTURER ON DIETETICS, GEORGE WASHINGTON UNIVERSITY; PATHOLOGIST, EPISCOPAL HOSPITAL; ATTENDING PHYSICIAN, FREEDMEN'S HOSPITAL, WASHINGTON, D. C.

From time immemorial it has been quite universally believed that during fever the digestive powers of the system are very greatly impaired, if not altogether abolished. In the standard medical literature of all ages, from Hippocrates to the present time, this doctrine is either explicitly stated or implicity conveyed. The belief has had a profound influence on medical practice, being one of the influential considerations leading to the low dietary regimen that has in all times almost universally prevailed in the treatment of acute diseases.

Although this belief still has a strong hold and strongly influences medical practice, it will, perhaps, not be unprofitable critically to consider its basis and validity, and to attempt to determine the exact condition and extent of the digestive processes and powers during fever.

On a general view, it seems almost self-evident that in such a profound disturbance of the body functions as occurs in fever, digestion must necessarily be enormously lowered. Such a belief accorded well with the views as to the nature of the febrile process that dominated medical thought throughout most of medical history. So plausible, indeed, is this belief, from the general conditions present in many cases of fever, that it is quite natural that it should have become a settled conviction; and strong evidence may justly be demanded to shake or modify it. Nevertheless, such a basis for this doctrine can be characterized only as a deductive generality, whose validity to be maintained must stand the test of a scientific and logical critique.

Clinical and general experience may be appealed to in support of the view of abolished digestion in fever. On its face this seems a consideration of the utmost weight, yet, as Hippocrates said, "experience is fallacious and judgment difficult," and the history of medicine shows that the appeal to the results of experience is an argument to be accepted with great caution. Many theories and systems and methods of practice for a time have enjoyed universal approval and claimed justification on the basis of general experience, which nevertheless in the end were found erroneous and were utterly discredited. Preconceived opinions are apt to interfere with unbiassed interpretation of results; and the occurrence of disturbed conditions in a few cases is apt to impress the

[1] Read before the Medical Society of the District of Columbia, October 19, 1910.

mind unduly, so that conditions that are really exceptional are taken to be the rule.

The usual occurrence of anorexia in fever is commonly regarded as a reason for withholding food. It should be noted, however, that loss of appetite does not necessarily imply loss of digestive power. Experiments with absolutely unpalatable and untempting diets (such as ash-free diets) have shown that such food is digested as completely as ordinary appetizing food (Pavlov's teachings to the contrary notwithstanding).

The dry mouth and tongue in many febrile cases have naturally suggested a diminution or suppression of salivary secretion and digestion in fever.

Numerous investigations of gastric digestion during fever have been made.

Beaumont from his classical researches (1833) claimed that in the "febrile diathesis" very little or no gastric juice is secreted and digestion is much impaired; the conceptions of fever at that time, however, were very vague, and it would appear from his protocols that his subject did not at any time during the experiments suffer from real fever, but only from transient attacks of acute indigestion.

Investigations by modern methods (*i. e.*, the stomach tube) have shown that in the various fevers there is usually a diminution or total suppression of hydrochloric acid in the gastric juice, while the pepsin is apparently but little reduced. Gastric absorption has been found materially retarded in fevers, while von Noorden found the motor functions of the stomach little impaired.

Special studies of pancreatic and intestinal digestion in fever have naturally not been made, owing to the inaccessibility of these regions to investigation.

Inasmuch as the bulk of digestion is carried on by the pancreatic and intestinal secretions, concerning which we have no specific data, it is apparent that the observations at hand indicating some lowering of gastric secretion are not sufficient basis for any reliable judgment as to the total impairment of digestion in fever.

Fortunately we have at command a simple, accurate, and conclusive means of determining the exact degree of the total net digestion and absorption of the various foodstuffs in patients with fever—the same standard method by which the quantitative digestion of food in health is determined—a method available and easily applicable for any subject. This consists in making exact determinations of the food materials (protein, fat, carbohydrate) ingested during a certain period, and also of these same materials as discharged in the feces for the same period. The difference between the material ingested and that dejected necessarily represents the amount digested and absorbed. The estimation of protein is based on a determination of the total nitrogen (Kjeldahl method)

in the food and feces, that of fat on the material extractible by ether. As a certain though indeterminate portion of the nitrogen and ether extract in the feces is derived from excretions into the alimentary canal, desquamated epithelium, bacteria, etc. (and not from the food), the digestion is always more complete than the figures indicate. The error thus introduced is of considerable magnitude with small diets, and should be discounted in interpreting such, but with ample diets is of smaller moment and does not vitiate the method for purposes of comparison.

In order to form an exact estimate of the degree of impairment of digestion during fever by this method, I have collected all the reports of analyses of food and feces in febrile diseases which I could find published. These cover 155 cases (1386 days of observation in all) in which determinations of nitrogen (protein) in food and feces were made, and 31 cases (154 days of observation) in which fat determinations were made. These estimations were made by thirty-six different investigators for various purposes. Practically no determinations of carbohydrate were found, and consequently there are no data on which to base a specific judgment as to its digestibility; but carbohydrate is usually digested much more completely than protein or fat, and it is highly probable that in fever the falling off in its digestion is no greater (and is probably less) than that of protein or fat. While there is ample room for further investigation, the data thus collected are sufficient for the formation of at least a tentative estimate of the impairment of digestion and absorption of protein and fat during fever.

The data are presented in the two accompanying tables, giving the general daily averages in food and feces during actual pyrexia of protein (nitrogen) and fat respectively. The actual diets in the various cases varied greatly both as to the amounts and the kinds of food used; many of the patients were on diets consisting largely or exclusively of milk, while many received a well-mixed diet. On account of the large error with small diets, cases in which the daily nitrogen intake was less than 5 grams have for the most part (except in children) been excluded from the compilation.

GENERAL AVERAGES PER DIEM OF DETERMINATIONS OF DIGESTION AND ABSORPTION OF PROTEIN (NITROGEN) DURING PERIODS OF FEVER IN VARIOUS DISEASES.

Disease.	Number of observers.	Number of cases studied.	Number of days of observation.	Nitrogen in food, daily average. Grams.	Nitrogen in feces, daily average. Grams.	Percentage of food protein (nitrogen) absorbed.
Typhoid fever	20	71	716	11.7	1.9	83.6
Typhus fever	2	15	84	9.9	1.4	85.7
Relapsing fever	1	1	6	7.6	1.1	86.2
Tuberculosis, pulmonary	11	33	323	21.0	2.6	88.3
"Chronic pneumonia" (probably including cases of phthisis)	3	6	79	11.8	1.4	87.8
Acute pneumonia	4	20	103	10.2	1.4	86.1
Acute pleuritis	2	2	14	9.8	1.9	80.6
Suppurative pyelonephritis	1	1	17	19.1	1.7	91.1
Scarlatina	1	6	44	5.3	0.6	88.6
Totals and general averages (by cases)	35	155	1386	13.3	1.9	86.0
Cases on exclusive milk diet		32	165	10.5	1.4	86.5

GENERAL AVERAGES PER DIEM OF DETERMINATIONS OF DIGESTION OF FAT DURING PERIODS OF FEVER IN VARIOUS DISEASES.

Disease	Number of observers.	Number of cases studied.	Number of days of observation.	Fat in food, daily average. Grams.	Fat in feces, daily average. Grams.	Percentage of food fat absorbed.
Typhoid fever	3	16	91	59.3	5.0	91.6
Typhus fever	1	4	18	23.4	3.9	83.2
Relapsing fever	1	2	8	25.8	4.4	83.1
Intermittent fever	1	1	6	19.4	2.6	86.6
Erysipelas	1	2	6	36.0	7.5	79.3
Tuberculosis, pulmonary	3	3	14	59.5	4.8	91.9
Acute pneumonia	1	2	8	22.3	4.7	78.9
Varioloid	1	1	3	43.4	5.7	86.9
Totals and general averages (by cases)	6	31	154	46.8	4.9	89.6
Cases on exclusive milk diet	2	22	84	38.4	4.6	88.0
Cases on diet of egg yolks	1	2	8	128.0	7.5	94.2
Cases on diet of ham	1	3	11	12.5	3.4	72.8
Cases on diet of porridge	1	2	8	84.0	7.1	91.6
Cases on mixed diet	5	8	43	73.9	6.0	91.9

For purposes of comparison the percentages of digestibility of the several foodstuffs in health, as determined by Atwater, may be stated, as follows:

	Protein.	Fat.	Carbohydrate.
Animal food	97	95	98
Vegetable food	84	90	97
Mixed diet	92	95	97

Examination of the figures in the tables shows for protein a general average digestion in fever of 86 per cent. in 155 cases of nine different diseases; this is a falling-off from the normal digestion of 11 per cent. if compared with animal food, or of 6 per cent. if compared with mixed diet, but is 2 per cent. better than the digestion of protein in vegetable food in health.

For fat, the general average digestion in fever in 31 cases of eight different diseases was 89.6 per cent., a falling off from health of 5.4 per cent. as compared with animal or mixed diet, and practically the same as the digestion of vegetable fat in health.

The determinations show no tendency for a lower degree of digestibility with the larger diets. In the typhoid fever cases, for example, the digestion and absorption of the food protein with varying amounts of protein in the daily ration was as follows:

Daily amount of protein in food.	Percentage of food protein absorbed.
Under 25 grams	57.9
25 to 49.9 grams	74.2
50 to 74.9 "	86.0
75 to 99.9 "	86.0
100 to 124.9 "	88.1
125 to 149.9 "	85.0
150 to 174.9 "	88.1
Over 175 grams	84.0

The cases on exclusive milk diet show a digestion of protein only 0.5 per cent. higher than the average, while that of fat is even less than the average. This showing does not support the prevalent belief in the superior digestibility of milk during fever.

In general, so far as shown by the data available, the average reduction of digestion during fever ranges not over 5 to 10 per cent. This falling off is so small as for practical purposes to be almost negligible; indeed, the digestion of animal and mixed food in fever is quite as good as that of vegetable food in health. The belief in a great impairment of digestion in fever is an idea based on vague impressions and dogmatic assumptions that has come down to us from the dark ages of medicine. The reduction actually shown by exact observations is so small as to dispel any fear, in general, of fever patients being unable to digest and absorb an adequate amount of the nutriment offered them, or to necessitate on that account any diminution of the diet.

REFERENCES.

Protein Digestion.

Abramovich, I. J. Thesis, Saint Petersburg, 1888.
Aikanoff, A. Thesis, Saint Petersburg, 1889.
Blumenfeld, F. Von Noorden's Beiträge zur Lehre vom Stoffwechsel des gesunden und kranken Menschen, Berlin, 1895, Heft 3, 101.
Bochkareff, I. E. Thesis, Saint Petersburg, 1893.
Bushuiev, B. T. Vrach, 1887, viii, 1003.
Diakonoff, D. I. Thesis, Saint Petersburg, 1890.
Grigoryeff, A. K. Thesis, Saint Petersburg, 1888.
Gruzdiev, B. C. Vrach, 1890, xi, 156, 178, 212, 257.
Huppert, H., and Riesell, A. Archiv der Heilkunde, 1869, x, 329.
Kornblum, H. Virchow's Archiv für pathologische Anatomie, etc., 1892, cxxvii, 434.
Kurloff, M. G. Thesis, Saint Petersburg, 1886.
Levin, A. M. Vrach, 1888, ix, 876.
Lipski, A. A. Vrach, 1888, ix, 643, 683.
Von Moraczewski, W. Zeitschrift für klinische Medicin, 1900, xxxix, 44.
Müller, F. Zeitschrift für klinische Medicin, 1887, xii, 87.
Pipping, W. Skandinavisches Archiv für Physiologie, 1891, ii, 89.
Sevastyanoff, A. T. Thesis, Saint Petersburg, 1891.
Walter, P. Thesis, Saint Petersburg, 1886.
Zasietzki, N. A. Thesis, Saint Petersburg, 1883; also, Virchow's Archiv für pathologische Anatomie, etc., 1883, xciv, 485.
For other references as to digestion in typhoid fever see Fiske Fund Prize Dissertation. Diet in Typhoid Fever, J. B. Nichols, 1907, p. 33.

Fat Digestion.

Blumenfeld. Loc. cit.
Chernoff, V. E. (Tschernoff, W.) Thesis, Saint Petersburg, 1883; also Virchow's Archiv für pathologische Anatomie, etc., 1884, xcviii, 231.
Von Hoesslin, H. Virchow's Archiv für pathologische Anatomie, etc., 1882, lxxxix, 95, 303.
Ladenburger, H. Thesis, Würzburg, 1901.
Lipski. Loc. cit.
Müller. Loc. cit.

HEMORRHAGES OCCURRING WITHIN A BRIEF PERIOD IN A GROUP OF CASES OF PULMONARY TUBERCULOSIS.*

By Charles M. Montgomery, M.D.,

INSTRUCTOR IN MEDICINE, UNIVERSITY OF PENNSYLVANIA; PHYSICIAN TO THE HOME FOR CONSUMPTIVES, CHESTNUT HILL, PA.

The rarity with which detailed reports have been published of hemorrhages in groups of cases of tuberculosis, and some features of special interest in the series herewith presented, are the occasion for the present report.

The outbreak occurred at the Home for Consumptives, at Chestnut Hill, Philadelphia, in which there were at the time 66 patients, 10 being incipient cases, 11 moderately advanced, and 45 far advanced. About one-fourth were males and three-

* Read before the Section on Medicine, College of Physicians of Philadelphia, November 28, 1910.

fourths females. Over one-half were between twenty and forty years of age. These figures, except that the total number of cases is a little low, correspond closely to the daily average at the Home. During the whole year 135 cases of pulmonary tuberculosis were under treatment, and during this time 43, or about one-third, expectorated blood at one time or another. The numbers expectorating blood each month were 11 in May, 15 in October, and in the remaining months varied from 2 in February to 9 in December. The longest period of absence from hemorrhage was sixteen days, from May 20 to June 4. There were two periods in which the hemorrhagic cases were concentrated, one from April 21 to May 19, in which 12 cases spat blood, and the period under consideration.

A glance at the table shows that in the brief period of three weeks, from September 30 to October 20, there were 14 individual cases expectorating blood, or between one-fifth and one-sixth of all the patients in the Home at the time, and about one-third of all the patients who expectorated blood throughout the entire year. On only one day of this period was there no patient spitting blood. During the previous three weeks only 4 cases and during the subsequent three weeks only 2 cases had hemorrhages. This is a large proportion of cases, even when compared to the frequency of hemorrhages in all cases of tuberculosis. This frequency varies, according to different statistics, from about one-fourth to over four-fifths of all cases, depending on the class of cases and the time they are under observation.

The patients at the Home are distributed pretty equally in four separate buildings, which are united by enclosed passages, and the number of hemorrhagic cases in this series was about equal in each building.

All of these 14 cases had expectorated blood at some time previously, but a considerable period had intervened in most of them, over three months in 5 of the cases; 1 incipient case was admitted during the outbreak and expectorated blood for the first time in a month.

The first case to develop hemoptysis during this outbreak was quite subject to hemorrhages. She had had streaky sputum a month before, and again two days before the first date given in the table, when she had a hemorrhage of eight ounces. Every day or two following for three weeks some new case would spit blood.

Besides the number of hemorrhagic cases in this series their severity is noteworthy; 6 of the 14 raised pure unmixed blood, and of these, 3 died immediately from the effects of the hemorrhage and a fourth expectorated twenty-four ounces of blood and died twenty-five days later without any recurrence of the hemorrhage. During the whole year 31 cases at the Home terminated fatally. Anders[1] records only 10 immediately fatal cases of hemorrhage out of 5302 cases. West[2] estimates that 1 to 2 per cent. of all deaths in phthisis are due to fatal hemorrhages.

No.	Sex.	Age.	Stage.	Condition prior to hemorrhage.	Sept. 30	Oct. 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
116	F.	23	F. A.	Progressive	8 oz.	..	..	5 oz.[1]																	
48	F.	29	F. A.	Stationary	..	B.S.	..	..	..	..	..	..	..	..	..	B.S.									
95	M.	46	F. A.	Progressive	..	B.S.																			
135	F.	18	F. A.	Improved	..	..	24 oz.[2]																		
160	M.	21	F. A.	Improved	..	..	..	1 oz.	B.S.	B.S.	B.S.	B.S.	B. S.												
12	F.	50	F. A.	Stationary	..	..	..	..	..	B.S.	B.S.	B.S.	B. S.												
17	F.	35	F. A.	Slightly prog.	..	..	..	..	..	..	..	1/16 oz.	1 oz.	4 oz.[1]											
16	F.	24	M. A.	Stationary	..	..	..	..	..	..	..	..	..	..	B.S.	B.S.									
6	F.	27	F. A.	Stationary	..	..	..	..	..	..	..	..	..	..	..	B.S.									
99	F.	22	F. A.	Stationary	..	..	..	..	..	..	..	..	..	..	..	..	2 oz.	6 oz.	1/8 oz.						
4	M.	62	F. A.	Stationary	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	B.S.	..	..	B.S.		
81	F.	29	F. A.	Progressive	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3 oz.	14 oz.	20 oz.[1]	
155	F.	35	M. A.	Stationary	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1/8 oz.	
166	F.	15	Inc.	Improved	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	B.S.

B. S. indicates blood-streaked sputum.
[1] Indicates immediately fatal hemorrhage (Nos. 116, 17, and 81).
[2] Patient died twenty-five days later, without recurrence of hemorrhage.

Even more remarkable are the number and severity of the hemorrhages in the female sex, all the six cases raising pure blood being females. The relative frequency of hemorrhages in males as compared to females has been variously estimated to be from a little less than 2 to 1 to a little over 3 to 1. Immediately fatal cases due to hemorrhage in females are considered to be very infrequent by Anders,[1] who was unable to find a single instance of fatal bleeding in 2453 female tuberculous cases, as compared to 10 fatal cases in 3420 males. Fatal hemorrhages at the Home for Consumptives have, as a rule, been of rather infrequent occurrence, only 1 case apart from the 3 here mentioned taking place within two years.

The immediately fatal cases were all far advanced, with extensive pulmonary lesions and cavity formation. No. 116 throughout her illness expectorated blood on nine occasions, exhibiting pure blood on six of these. No. 17 had a total of fourteen hemorrhages, of which thirteen consisted of pure blood. No. 81 had a total of thirteen hemorrhages, twelve of pure blood.

In one of the fatal cases the bleeding was associated with the menstrual period, half a dram of blood being expectorated on the day after the onset of the menses, one ounce on the following day, and on the next day the fatal hemorrhage occurring. It is by no means rare for hemorrhages to occur at the menstrual periods, in some cases recurring with a certain degree of regularity, and in fact the last hemorrhage this patient had been seized with ten months previously occurred synchronously with her menses. However, I have not met with a report of any other case in which a fatal hemorrhage took place at this time. Subsequently B. I. Macht[3] mentions a fatal case reported by Flesch.

The amount of the hemoptysis gives no definite indication of the quantity of blood lost or of the serious effects produced. This is well illustrated by the immediately fatal cases referred to in the table. Osler mentions a case in which the bleeding was fatal without the appearance of any hemoptysis, an eroded vessel having opened into a large cavity. The immediate effects of a hemorrhage will depend on the amount of blood lost, on the degree of suffocation, or on both.

How is the occurrence of hemorrhages in groups of cases to be explained? So many and so severe cases as this series presents can hardly have been the result of a mere coincidence, and one looks for some common cause or causes to explain the whole group.

Exertion, excitement, etc., may be excluded as causes in this outbreak, as all the patients were leading quiet lives, all the ones with fatal hemorrhages having been in bed for some time previously, and some of the severe hemorrhages having occurred at night. In fact, I can recall no case in which a hemorrhage at the Home could be traced to exertion, and many of them have taken place at night.

Seasonal and atmospheric conditions have been offered as causes of hemorrhages. On this supposition two explanations suggest themselves: (1) that these conditions affect the foci of disease, thereby producing hemorrhages; and (2) that they in some way affect pathogenic bacteria or render their hosts more susceptible, the resulting activity of the germs causing hemorrhages. Colds, bronchitis, pneumonia, and even the hemoptysis of phthisis have been stated to be more frequent in winter and spring than in summer and early fall.

West, however, finds in statistics no proof of any seasonal influence on the occurrence of hemoptysis. The present outbreak occurred at a time of year generally considered to be pretty healthful. In the year under consideration the rainfall and temperature were both below the average for this particular period.

Minor[4] states that "he has noticed that close, damp, hot spells, with a low barometer, seem to be the time when many cases of hemorrhages are apt to occur, and that this bunching is much less common in winter. Thus, in the practice of Dr. W. L. Dunn, in the summer of 1907, sixteen hemorrhages occurred among his patients in one week."

Of the various explanations offered to account for these hemorrhagic outbreaks, the most reasonable seems to be that some of them are due, at least in part, to an acutely infectious origin. Such a conclusion, however, is drawn much more from inference than from definite scientific data. An infectious origin is suggested (1) by the grouping of the cases; (2) by the evidences of toxemia, such as fever and pain; (3) by the development of definite acute infections in some of the cases of hemorrhage, for example an acute pneumonia; (4) by the occurrence at the same time in other patients of symptoms of acute infections like acute colds; and (5) by bacteriological examination of the sputum and lungs in a limited number of cases.

As to the first point, this series of cases is simply an exaggeration of what is constantly being encountered in institutions. Indeed, so frequently do hemorrhages occur in groups of cases that one is disposed to regard the appearance of one case as the probable herald of more to come.

A rise in temperature, both in those patients spitting pure blood and those with only streaky sputum, occurs sufficiently often to suggest an acute infection. However, such a rise is frequently absent, and among our 14 cases occurred in only three. This absence of fever is not surprising if one supposes that the causative agent consists in the microörganisms of acute intercurrent infections, like acute colds, which in normal persons may cause hardly any rise of temperature. Many tuberculous patients already have some fever, while advanced cases often show very little tendency to febrile reactions.

The records as to pain are too few in the present series to be of value, but pain is a frequent accompaniment of hemorrhage from the lungs. The pain is more apt to be localized to some spot or area of the chest than to be distributed over the body, thus differing from the general pain often encountered in the toxemias of acute infections in non-tuberculous individuals. West[5] has called attention to the pain preceding hemorrhage and relieved by it. He attributes the pain to a more or less sudden congestion. Wood[6] believes that evidences of pleuritis in phthisis may be due to a toxemia from infections of the upper respiratory tract, the pleura in many of these cases being a point of lessened resistance. It seems quite possible that toxemia may at times cause congestion, which in turn may produce both pain and hemorrhage. Localized tenderness of the chest is another frequent accompaniment of hemorrhage. I[7] have reported 8 cases of pain among 10 cases with hemorrhage, and 10 cases of tenderness among 11 cases with hemorrhages.

The theory of an infectious origin is further supported by the symptoms suggesting an acute bronchitis, such as increased cough and expectoration, tightness or a sense of oppression in the chest, and dyspnea. Patients are frequently forwarned of hemorrhage. and West records a patient who could always foretell the onset of a hemorrhage by the pain in the chest. Moreover, at the time of these outbreaks other patients may be suffering from acute colds, though not themselves spitting blood.

The frequent occurrence of small amounts of blood in acute infections of both the upper and lower respiratory tract even in the absence of tuberculosis is significant. McPhedran[8] states that the bacteria most often found in acute bronchitis, when not due to specific infections, like influenza, pertussis, typhoid etc., are the pneumococcus and streptococcus, which may occur singly, in association with each other or with other bacteria. Osler[9] mentions a case of hemorrhage in typhoid in which the lungs at autopsy revealed no lesion. Flick, Ravenel, and Irwin[10] have found in the sputum and lungs from hemorrhagic tuberculous cases a number of organisms, but attributed most importance to the pneumococcus, being impressed by the association of pneumococci and hemorrhages in cases of pneumonia.

It would be valuable to know whether acute intercurrent affections of the respiratory tract occur less at some localities than others, and whether there is a corresponding decrease in the number of hemorrhages. The more isolated the sanatoria the more infrequent one would expect to find these acute infections. At Fort Stanton, New Mexico, Smith[11] did not observe any grouping of the cases of hemorrhage (cases with only streaky sputum being excluded), nor any special association between hemorrhages and the humidity or barometric pressure.

Undoubted evidences of acute infection are at times encountered

even when the hemorrhage is large. Sudden high fever, consolidation in the lung, and death may occur within a few days or weeks after a hemorrhage. While in some of these cases infection follows insufflation of blood, in others the infection is primary, and the hemorrhage is a result.

Considerations like the preceding with all their deficiencies and inconsistencies lend weight to the idea that a certain number of hemorrhages may be due, at least in part, to prevalent, that is, contagious intercurrent affections, probably including the common colds. It is natural to suppose that the resulting congestion would particularly affect tissue already injured by disease.

While we often cannot differentiate by means of the symptoms and physical signs between renewed activity of the tuberculous process and some other process—for instance, some intercurrent affection—we may assume that in a certain number of cases only the latter has been in progress, when the symptoms and signs are slight and disappear in a short time without leaving any appreciable traces of their action. Thus blood-streaked sputum, and at times quite large hemorrhages, occur in apparently healed cases of tuberculosis, being accompanied by symptoms of an acute infection, which soon pass off, leaving the patient as well as before.*

Just what all the operative factors may be in the diseased focus at the time of a hemorrhage cannot be determined. In those cases in which we assume there is no renewed activity on the part of the tubercle bacillus or fresh output of tuberculous toxins, the focal phenomena cannot be specific in character, only the toxins of the tubercle bacillus and not those of other bacteria having any specific action on tubercular foci (Denys[12]). Perhaps there is sufficient congestion in some of the cases to excite hemorrhage in a focus already diseased, and containing bloodvessels with weakened walls, so that it apparently offers less resistance to even purely mechanical factors, such as increased blood pressure. In a similar way increased intravascular tension may be supposed to act upon a tuberculous area in cases of menstrual hemoptysis, the focus of disease, however, revealing no evidences of activity at the time of the menses.

On the other hand, if increased blood pressure at the focus of disease is the only exciting cause of these hemorrhages, why does not violent exertion more frequently cause them? With sudden exertion the rise in pressure comes on much more abruptly even if it is less sustained than in acute infections or during the menses. Therefore, in the latter conditions some factor or factors additional to the increased blood pressure would seem to be operative. The circulation in the blood during an acute infection or during menstruation of substances that have some effect on diseased tissue

* In the discussion Dr. Gwyn referred to a case of apparently healed tuberculosis that had a hemorrhage during an attack of mumps, no evil consequences following.

predisposing it to hemorrhage may possibly be a partial explanation, without, of course, having any data in its support.

REFERNCES.

1. Anders, J. M. Jour. Amer. Med. Assoc., liii, 455.
2. West, S. Diseases of the Organs of Respiration, 1902, i, 382.
3. Macht, B. I. Amer. Jour. Med. Sci., 1910, cxl, 852.
4. Minor, C. L. Klebs, Tuberculosis, 1909, p. 214.
5. West, S. St. Bartholomew's Hospital Reports, 1882, xviii, 263.
6. Wood, G. B. Jour. Amer. Med. Assoc., lii, 1063, ab. Annals of Otology, Rhinology, and Laryngology, St. Louis, December, 1908.
7. Montgomery, C. M. Tenderness in Pulmonary Tuberculosis, Henry Phipps Institute, Fifth Annual Report.
8. McPhedran, A. Osler's Modern Medicine, iii, 636.
9. Osler, W. Practice of Medicine, 1896, p. 26.
10. Flick, Ravenel, and Irwin. Medical News, New York, September 9, 1905.
11. Smith, F. C. National Association for the Study and Prevention of Tuberculosis, Fourth Annual Report.
12. Denys, J. Sixth International Congress on Tuberculosis, vol. i, pt. 2, p. 751.

MOUNTAIN SICKNESS.

By August Strauch, M.D.,

CHICAGO, ILLINOIS.

Travelling and sojourning among mountains is a prominent feature of modern life, because of the increasing enthusiasm for Nature's grandeur and the growing appreciation of the hygienic factors of the climate of high altitudes by the healthy as well as by the sick. The number of resorts and sanatoria in the regions of high mountains is ever increasing, to combat various diseases, successfully, as the empiricism of centuries has proved.

But as the advances became bolder, penetrating into the highest altitudes, partly for touristic purposes, partly for scientific exploration and research, and as mountain wandering became more common, due to successful geographical exploration and development of railways, that added to the accessibility of the higher mountain systems, experiences accumulated, that pointed to harmful effects and dangers from the climate of high altitudes above a certain zone. The study of these effects, that high altitudes exert upon the human organism in a physiological and pathological sense, is highly interesting from a scientific point of view; it also has an important bearing upon the practical question. How may the hygienic factors of high lands be utilized to the best advantage by both the sick and the healthy, and what are the indications and contraindications for high altitudes?

The increasing numbers of Americans that resort to the European Alps and to the high elevations of the Rocky Mountains in our Western States make this subject one of concern to all physicians.

The Federal Government of Switzerland considered it of great practical importance to ascertain, when the construction of mountain railways to the highest summits of the European Alps was contemplated, where the danger line was, at what altitudes injuries to the health of visitors might presumably be inflicted, and which individuals might be seriously imperiled. In connection with these studies, Kronecker's account of his observations in the pneumatic chamber, and on mountains, is one of the most important works written on mountain sickness. But also reports were then available of experiences made on the high mountain railroads of the American continent, especially on the famous Oroya Railroad in the Peruvian Cordillera, the highest railroad of the earth, reaching the considerable elevation of 15,800 feet, and also of experiences on the cog-wheel railroad to the summit of Pike's Peak, 14,200 feet.

Historical Remarks. The exploration and conquest of the high European summits had not yet commenced when the Spaniards, on their conquering expeditions into the equinoxial regions of ancient American civilization, penetrated into the mountain chains of the Great Andes, and advanced to altitudes where they could not escape the serious effect of the rare climate upon the health. Numerous are the records of this period of conquest, vividly narrating the great agonies and miseries which the bold pioneers had to endure on the high passes of the great Andes.

Perhaps the most interesting are those of the Jesuit, Pater Acosta, who, as the first one in his *Historia naturale de las Indies*, gives us a detailed account of the symptom complex which he named mountain sickness.

On his expedition to Peru, at the altitude of about 14,800 feet, when passing the Periacaca on his mule, he was seized by an attack of discomfort, with abdominal cramps and the vomiting of food, mucus, and bile, and finally blood; his companions suffered from similar attacks, and also from diarrhea. It deserves to be mentioned that he ascribed these symptoms to the rarity of the atmosphere.

Neglecting a few records from the following period, we see their number rapidly grow at the end of the eighteenth century, the beginning of the period of great scientific explorations of the higher mountain systems of our earth, relative to their geography, orography, geology, etc.

De Saussure relates that, during his famous expedition on the summit of Mont Blanc in 1787, an overwhelming sleepiness, which defied cold, wind, and snow; lethargy, lassitude, vanishing of muscular power, and vomiting was experienced among the participants of the toilsome ascent. He had to take breath every

ten to fifteen steps, being compelled by exhaustion to sit down. When trying to rise up again his legs failed. The fatigue, which he explained by rarity of air, was overwhelming. He suffered from intense heart palpitation, beating of the arteries of the head, much dyspnea, a feeling of faintness, and complete loss of appetite.

In his records he at the same time points to the remarkably speedy recovery from the exhaustion after a short rest of two to three minutes, so differing from the symptoms of exhaustion in low altitudes due to actual overexertion.

On the summit of Mont Blanc, 15,800 feet, where he made meteorogical observations, he suffered at rest only from slight nausea, but every attempt at reading his recording instruments in the rarefied air was followed by intensive dyspnea as a consequence of the short suspension of respiration due to the intentness required for the observation.

In the beginning of the nineteenth century, Alexander von Humboldt[4] undertook his well-known scientific expeditions to Central and South America, where he explored the great mountain chain of the Cordillera, in its average height, the second on earth. Thrillingly he depicts the sufferings which befell him at the altitude of about 5800 meters (19,000 feet), during his ascent of the Chimborazo volcano, that, towering with its five summits, reaches the height of 6250 meters (20,600 feet).

"The Indians, with one exception, abandoned us at an altitude of 15,600 feet. Prayers and threats were alike vain; they declared they were suffering more than we. When the height of 5800 meters (19,000 feet) was reached, all of us by degrees began to feel very ill at ease and to suffer from great nausea; the vomiting was accompanied by dizziness, and was more distressing than the difficulty of breathing. The companions bled from gums and lips, and the conjunctivas of their eyes likewise were bloodshot."

Once when upon the Pinchincha, at an elevation of 13,800 feet, he was seized with such violent pain in the stomach and overpowering giddiness that he sank upon the ground in a state of insensibility.

Vivid pictures of self-experienced sufferings during travels among the great Andes we owe also to Poeppig, of Leipzig, Tschudi, Whymper, Conway, and others.

Tschudi,[13] who travelled in Peru from 1838 to 1842 (*Travels in the Great Andes*, translation from the German), depicts one of his attacks of mountain sickness at an altitude of about 4500 meters (14,800 feet) in the Cordillera.

"My panting mule slackened his pace and seemed unwilling to mount a rather steep ascent. To relieve him I dismounted and began walking at a rapid pace. But I soon felt the influence of the rarefied atmosphere, and I experienced an oppressive sensation, which I had never known before. I stood still for a few moments

to recover myself and then tried to advance; but an indescribable oppression overcame me. My heart throbbed audibly, my breathing was short and interrupted. A world's weight seemed to lie upon my chest; my lips swelled and burst; the capillary vessels of my eyelids gave way, and blood flowed from them. In a few moments my senses began to leave me. I could neither see, hear, nor feel distinctly. A gray mist floated before my eyes. In this half senseless state I lay stretched on the ground until I felt sufficiently recovered to remount my mule."

Dwelling upon the subject of mountain sickness, he mentions the fact, that high altitudes are very fatal to cats. At the elevation of 18,000 feet above the sea, these animals cannot live; numerous trials have been made to rear them in the villages of the upper mountains, but without effect, for they die soon here under convulsions. Horses and mules suffer like man.

It is only more lately that experiences as to mountain sickness in the great mountain systems of other geographical positions have been gathered, a fact which may be explained by the circumstance that the great Andes of Peru and Ecuador are only to a small extent covered with eternal snow and glaciers, on account of their equatorial latitude, and for that reason they have been inhabited from time immemorial to very considerable elevations, and, therefore, are comparatively easy of access.

The temperate climate renders it possible, as high as Quito, 2850 meters (9400 feet), for bananas, palms, and eucalyptus to grow on protected places, and an abundant flora of alpine flowers ascends to an elevation of 4000 to 4800 meters (13,000 to 15,800 feet).

Thus, numerous records have been made of observations on mountain sickness during various ascents of the summits of the Caucasus, the Ararat, 5200 meters (17,000 feet), of Mount Loa, 4170 meters (13,760 feet), etc., and finally during travels on the great Central-Asiatic high plateaus of Thibet, and in the Himalayas (Sven Hedin, Guillermod, Kronecker, and others).

The authors emphasize the fact that the higher the altitudes the greater is the difficulty of walking and breathing, and that physical exertion even of a minor degree augments the suffering in an extraordinary way.

The brothers Schlagintweit, well possessed of an extraordinary resistance against the air of high altitudes, who on their scientific mission to high Asia succeeded in carrying their climbing as high as 6800 meters (22,000 feet), corroborate the fact that the native Hindoo coolies, the carriers of burdens, are often more affected than the European travellers who are riding. "Our guides and servants," he states, "dropped upon the snow, declaring they would rather die than take one step more."

Whymper,[14] the well-known English high-tourist, who, among

mountain climbers, has devoted more particular attention to mountain sickness, experienced manifestations of mountain sickness, as related in his work on the Chimborazo in the region of 16,000 feet above the sea, corresponding to a barometric pressure of 400 mm. After a meal he and his two companions were suddenly seized with an alarming attack of the malady, with great weakness, fatigue, thirst, headache, a feeling of fever, extreme dyspnea, heart palpitation, and acceleration of the pulse. He adds this remark: "Our incapacity was neither due to exhaustion nor to deficiency of bodily strength, but to the rarity of air."

Definition and Symptoms During Work. Scrutinizing the accounts of pathological phenomena that occur during travels in high mountains, in order to define the clinical entity of the mountain sickness proper, we ought to regard only manifestations that in a direct or specific way are due to the high elevation itself, and we must subtract those that may come from causes also operative in lowlands, that is, symptoms of an actual overexertion, snow blindness, erythema solare, etc.

The mountain sickness befalls some individuals at a lower, others at a higher altitude, but no one who proceeds beyond a certain elevation, the critical line, escapes the malady. An elevation of 3000 meters (10,000 feet), or even less, might provoke it in some, others may escape the symptoms up to 4000 meters (13,000 feet), or even 5000 meters (16,500 feet), while only very few, possessed of unusual resisting power, can without much distress venture upward to 6000 or almost to 7000 meters (19,000 to 23,000 feet).

The symptoms of mountain sickness depend not only on the nature of the individual, but also on various intricate contingencies, especially on physical exertion, that is, on whether the ascent is performed by climbing or by passive carriage on horseback, sedan chair, or mountain railroad.

In the former case, as might be expected, the symptoms make their appearance in lower altitudes, and involve those organs which are under the greatest strain, namely, the heart and muscles. Marching or climbing within the critical zone causes fatigue, faintness, and physical prostration, that often set in rather suddenly and after even a comparatively small amount of physical work; they may be so severe as to necessitate rest, or even to lie down; the failure of the muscles to act may completely incapacitate the traveller for movement. But in not extreme elevations, a few moments of repose apparently restore the former strength, and with new muscular vigor one takes up again his climbing, to be forced, however, to halt anew after perhaps only a few steps.

The great intensity of the fatigue, the readiness with which it appears upon even moderate muscular activity, which in low altitudes hardly would have any perceptible effect, distinguish it strikingly from ordinary fatigue, called forth by actual overexertion.

The strange feeling of faintness and the quick exhaustibility in mountain sickness, therefore, is not identical with ordinary exhaustion by excessive muscular work, but it is generally admitted to be due to the deficiency of oxygen in the rarified air, that cannot satisfy the body's increased demand on account of the increased work of the heart and muscles.

But speedy reparation takes place for when at rest, the consumption of oxygen falls off rapidly.

To the above-mentioned fatigue and weakness are added heart palpitation, dyspnea, oppression of the chest, vertigo, anorexia, headache, often ringing in the ears and impairment of hearing, nausea, vomiting, great drowsiness, and sometimes syncopal attacks.

That cases of genuine overexertion with the well-known trail of serious symptoms, such as acute dilatation and insufficiency of the heart, feeble dicrotic "fatigue" pulse, etc., are encountered in mountains more frequently than in lowlands is not to be wondered at, as mountaineering represents intense muscular work, often carried beyond the limits of endurance and capacity for muscular exertion, in the case of untrained summer tourists, either from necessity or temptation.

But these symptoms are not from mountain sickness primarily, being similar to those occurring in overexertion in lowlands; though more grave significance is attached to them on account of the rarity of oxygen in higher altitudes. Recovery takes place more slowly and exitus lethalis is more liable to be the issue.

In pronounced cases of mountain sickness, as, for instance, in very high altitudes, psychical manifestations may supervene. The will power is then extinguished, mental lassitude prevails, and in consequence a complete indifference toward the dangers of snow, cold, storm, etc., gets hold of the individual, "who would rather die on the spot than make an attempt to proceed."

States of mental excitability, as well as states of physical depression, are encountered; increased liability of the emotions tending to quarrelsomeness or hilarious expansivity. This is well known to the guides and tourists of the Alps. Cyanosis, blue circles around the eyes, bleeding from nose, lips, gums, lungs, and stomach, and constipation or diarrhea, are other symptoms referred to in the records on mountain sickness.

Author's Experience. I experienced the effect which comparatively moderate altitudes exert, in tending to lower physical endurance, on the high plateau of the Yellowstone geysers, the elevation of which is about 7000 feet, where fast marching or, more markedly, running on a horizontal level for a short distance such as 300 to 350 yards, called forth a very high pulse rate (150 to 160) and dyspnea, that I never experienced at lower levels with such insignificant muscular work. Many tourists, although trained in various sports,

expressed their wonderment at how quickly fatigue arose when they wandered on the fairly horizontal plateau of the geysers.

When I had reached the critical line, in ascending the volcano Etna (10,800 feet), at an altitude of about 9600 feet, the rarefied atmosphere made itself felt to me in a considerable degree; in spite of a good night's rest in the observatory at the base of the central cinder cone (9650 feet), the final stage of the ascent was made under great fatigue, heart palpitation, very high pulse rate and difficulty of breathing, so that I was forced to halt every fifteen to twenty-five steps, but restoration of apparently full muscular strength promptly occurred after a rest of only one to two minutes. It was obvious that exhaustion due to actual overexertion was out of the question, for the smooth cinder sides of the cone, inclined at a slope of about 30 degrees, were rather compact on account of previous rains, so offering a splendid foothold. On the top, at rest, I felt perfectly well.

Perhaps tourists here show manifest symptoms of mountain sickness, such as lassitude, headache, apathy, nausea, vomiting and dyspnea at the slightest movement, etc., more frequently than on other mountains of the same altitude. This is possibly due to the fact that the ascent begins practically at sea level, and is performed without intermediary stages.

Symptoms of Mountain Sickness Without the Influence of Exertion. As the influence of the muscular exertion of climbing, or of occasional actual overexertion, renders the clinical picture more complex, and, therefore, rather difficult of analysis, we may study with great advantage the syndrome that arises when a high altitude is reached passively, that is, without physical work, as by mountain railroad, riding on horseback, or by ascending in a balloon. Here symptoms are forthcoming that at first point to disturbances of the brain functions; such as headache, dizziness, trembling, apathy, lassitude, and sleepiness, to which are added general weakness, often tinnitus aurium, nausea, and vomiting, sometimes obscuration of the vision and, if aggravated, even syncope.

Moderate exertion, as wandering on a level, or to a greater degree climbing, increases the manifestations, or if they have not been developed, calls them forth; dyspnea sets in, and the pulse quickly rises to a very high rate. The disturbances during high balloon ascensions are similar. However, the symptoms mentioned, as a rule, do not appear immediately after the arrival on the height, but may develop gradually, that is, within hours, the speed of the passive ascent having an important bearing. Complete loss of appetite also prevails; thus, the whole picture resembles the state of acute anemia.

Author's Experience on Pike's Peak. In 1908 I made an ascent of Pike's Peak (14,200 feet) by the cog-wheel railroad. The trip to the summit took one and one-half hours. After arriving, about noon, my pulse rate at rest was increased to 70, the respirations

to 30. Attacks of dizziness, sensations in the ear, tremor of the hands, and a desire of more frequent and deeper respiration, often with sighing, were the clinical manifestations. Moreover, dyspnea and dizziness became aggravated and the pulse rate very much increased by ridiculously insignificant exertions, as by leaping over the granite blocks on the summit, by fast walking, and especially by stooping; I was unable to tie my shoes without pausing in between to gasp for air; and oftentimes I had pulsations in the eyes. I passed in bed an entirely sleepless and miserable night, on account of malaise and attacks of slight oppression of the chest. The pulse rate was between 90 and 100, the higher rates after slight movements. Most of the night there was a pronounced periodical type of breathing, approaching Cheyne-Stokes' phenomenon, namely, long pauses, during which my drowsiness increased, were followed by a series of a few very deep inspirations, which in their succession gradually became superficial ones.

The following day the attacks of dizziness and the pulsations in the bulbus had improved.

The lowest altitude in Colorado at which I experienced dizziness, that at the same time caused uncertainty in walking, was at 10,800 feet, when going over Marshall Pass by railroad.

Under physiological conditions in low land indications of a periodical respiration can only be observed, during sleep, when the depth of the respiratory movements, gradually increasing and decreasing, forms thus connected groups.

This physiological phenomenon is explained by the assumption of a lowered irritability of the nervous respiratory centre during sleep. In high altitudes, however, this periodicity is more pronounced, and the respiratory curves are also undulating while awake. Mosso observed it on the summit of Monte Rosa (15,300 feet) in almost everybody.

The types of the respiratory periodicity in high elevations are various, as illustrated in Mosso's monograph,[9] and include also characteristic Cheyne-Stokes' phenomenon. The occurrence of the latter in very high altitudes, however, has not the ominous significance that otherwise is attached to it.

Prognosis. Although the malady may have aggravated manifestations with great discomfort and distress, in moderate altitudes these, with time, especially when at rest, tend to diminish rather than to increase, and the danger to those otherwise healthy, is generally slight. However, a few cases of exitus lethalis are ascribed to the direct effect of the rarefied atmosphere; the French scientists Sivel and Crocé-Spinelli, lost their lives in their balloon at an altitude of about 8600 meters, while Tissandier, the third participant of the ascension, fell unconscious and narrowly escaped.

Accommodation and Acclimatization. As a rule, in the course of days or weeks, a certain degree of acclimatization takes place.

The laborers occupied with the boring of the Mont Blanc tunnel suffered only in the first weeks from short breathing, drowsiness, loss of appetite, and great exhaustibility.

There are many permanently inhabited places on the slopes and high plateaus of the Andes up to the considerable elevations of 13,000 to 15,000 feet; and the mines here of Villacota, situated at an altitude of 5000 meters (16,500 feet) are operated by indigenous and foreign laborers. The natives of the places are capable of speedy marching in heights where newcomers from Lima suffer great distress.

In Quito, 2850 meters (9400 feet), a city of 50,000 inhabitants, bull-fights are performed, and in Potosi, 4000 meters (13,200 feet), "the girls can dance throughout the night as in our country."

However, it must be mentioned that the adaptibility of the human organism is limited, the acclimatization being effective only in those altitudes in which one stays permanently, while failing completely in extreme heights as at 6000 meters (19,800 feet).

Causes. The physiological and pathological manifestations observed in high altitudes, as described, can experimentally be produced by gradually rarefying the atmosphere within a pneumatic chamber, imitating thus the decrease of the barometric pressure and of the amount of oxygen with the increasing altitude. Such experiments, "artificial ascents," under various conditions, have been made by Paul Bert,[7] Mosso,[8] Reynard, Schroetter, Zuntz, Löwy, and others on men and animals, as on monkeys, cats, dogs, guinea-pigs, birds, etc., to elucidate the etiology and pathogenesis of mountain sickness.

As the air in the chamber gets decompressed, the individual in lying posture first notices, perhaps, a slight feeling of pressure of the head, buzzing in the ears, and an increasing tiredness and drowsiness, which with the proceeding diminution of the air, more and more becomes irresistible. Slight exertion, for instance, sitting up, handling apparatus, or stooping, aggravates the fatigue and faintness considerably, the muscular strength fails, dizziness and ataxia occur, the eyesight is dimmed, the psychical functions—for instance, the memory, attentiveness, concentration, etc.—are impaired, and headache, dyspnea, heart palpitation, sometimes also nausea, manifest themselves. After lying down again, or by inhalation of oxygen, the symptoms improve or may disappear entirely.

Animals get less lively, motionless, often vomit, and fall into a doze or unconsciousness.

The muscular ataxia in much rarefied air shows itself also in alterations of the handwriting. Of this, H. v. Schroetter, at an air pressure of 240 mm., after a short interruption of oxygen inhalation, has given a striking example; his hand became hardly legible.

Zuntz[17] reports the observation of similar alterations of the hand-

writing of Caspari and Mueller on the summit of Monte Rosa, 4650 meters (15,300 feet), and, according to him, very marked is the loss of control of the voluntary muscles shown in the meteorological notes of Berson and Suering[1] during their well-known balloon ascent in 1901, whereby they reached the highest altitude as yet obtained by man—10,800 meters (35,600 feet).

The theory at the present time accepted by most investigators as to the causation of the malady in high altitudes holds the rarity of the oxygen in the atmosphere responsible. It causes a diminution of oxygen in the blood, a state of "anoxhemia barometrica," as Jourdanet, the founder of this theory, has termed it, and consequently a kind of asphyxia of the tissues.

Inhalations of oxygen in the pneumatic chamber and in the balloon have a beneficial effect, retarding or relieving the symptoms, and rendering man capable of withstanding degress of air rarity or altitudes which otherwise would be fatal.

During high ascensions, after the first draughts of oxygen the strength of aëronauts returns, fainting fits disappear, the beginning psychic lethargy changes to clear consciousness, physical work can be done again, etc.

With oxygen inspiration, Paul Bert[2] could submit himself in a metal cylinder to a decompression of the atmosphere which corresponded to altitudes of the summit of Chimborazo and of Mount Everet, the highest mountain on earth.

Mosso, in his experiments, endured thus an air pressure corresponding to an altitude of 10,500 meters (34,600 feet), and Suering in his balloon ascension could with oxygen supply make the record of an elevation of 35,600 feet, the highest as yet obtained.

In connection with such facts arises the question, interesting from a physiological and biological standpoint, as to the maximum altitude compatible with human life.

H. v. Schroetter[12] (Vienna), by calculation, places the limit at about 12,000 meters (39,000 feet), for here even inhalation of pure oxygen would be without avail, because at such an altitude the partial pressure of oxygen in the lung alveoles would be too low to allow its absorption.

The great exhaustibility by muscular work in high mountains has its analogy also in the experiments of Reynard, 1894, who found that a guinea-pig, forced to move about within the recipient of an air pump, at an air pressure corresponding to 4600 meters (15,000 feet) altitude, very soon became exhausted and fell, while another guinea-pig, left at rest, fell and seemed to die only at an air pressure which corresponded to an altitude of 8000 meters (26,400 feet). When air was quickly admitted, both animals recovered, the first one, however, very slowly.

Anoxyhemia gives a satisfactory explanation for the fact that mountain sickness appears sooner during muscular work than at

rest, as during ordinary mountain climbing the demand for oxygen is increased four to five times more than the amount demanded during rest. Therefore altitudes such as have been reached in balloons can hardly be obtained by climbing, and the climbing up to almost 7000 meters (23,000 feet) in the Andes and Himalayas, as done by Conway,[3] Zurbrigger, and Stuart Vines, although with difficulties, must be considered as an extraordinary feat and as a proof of a very rare resistive power.

The accumulation of pathological products of metabolism, that gradually develop on account of the insufficient oxygen supply in the blood and betray themselves not only in the increased output of incompletely oxidized substances in the urine, but also by the decrease of the alkalinity of the blood (Zuntz), may account for the fact that the effects of high altitudes often are cumulative, the manifestations often appearing not immediately upon arrival on the summit, but gradually within hours, or even posthumous, so to speak, during the descent.

The acclimatization to high altitudes is principally due to an increase in the total amount of hemoglobin as the oxygen carrier, and to an alteration of the respiration and circulation, in a regulative sense.

As a matter of fact, the natives of the high plateaus of the Andes show a remarkably higher number of erythrocytes than the inhabitants of lowlands, namely, 6,000,000 to 8,000,000 in 1 c.mm.; a gradual increase of the number of red blood cells is also observed in man and animals, who temporarily remain in high altitudes.

Paul Bert, in 1882, found that the blood of the Peruvian mountain sheep (llama) was capable of absorbing much more oxygen than was the blood of sheep living on the plains.

Miescher[7] suggested, and Zuntz and Löwy proved by experiments on animals, that the air of high elevations stimulates the function of the red bone marrow; the total amount of blood and hemoglobin which could be extracted from the body being acually increased.

Jaquet[6] obtained quite similar results by keepting rabbits in chambers in which the atmospheric pressure was artificially reduced.

Humboldt found a remarkable increase in the capacity of the thorax among the inhabitants of the great Andes, and Dr. C. T. Williams[15] likewise observed an actual increase in the thorax as the result of a residence in high mountain resorts.

Besides the diminution of oxygen in high mountains, some accessory factors, the sum total of the other climatic factors, including the minute electric conditions of the atmosphere, seem to have a certain bearing upon the outbreak of the malady, although not yet well understood. Intensive cold temperatures, dry air, clear skies, when, as it is found, the ionization of the atmosphere is increased, and also strong sunshine are, according to the general experience, largely contributory to the provocation or aggravation

of the symptoms, while vaporous air, cloudy skies, and winds tend to retard or lessen them. A very frequent statement is made that during the night's repose the symptoms become more distressing. Likewise very remarkable is the fact that the symptoms of mountain sickness may appear especially on certain locations of predilection, as is well known on the Alps and the Andes, though other regions of the same mountain range, or of the same mountain even situated higher, may allow mountaineering without suffering. Such places of predilection often are narrow couloirs, ravines, trough-shaped depressions of the niveau, in short, locations more or less protected from the motions of the air, and, therefore, not ventilated. On such places, according to Elster, Geitel, and Zunts, a high ionization, unipolarity, and radio-activity of the air exist as the result of the stagnation of the radio-active emanations from the soil. Saake[16] found the air of high altitudes to possess three times the amount of radio-active emanations than the air of lowlands, and it is known that certain soils and minerals, for instance, those containing pitch-blende or some other metal ores, some clays, etc., emit a greater amount of emanations than others. But further investigation and advance of our knowledge of the influences which the minute electrical and other physical conditions of the atmosphere exert upon the human body, as yet hypothetical, will be necessary before we can pass on the problems that the above-mentioned facts involve. Obscurity prevails also in regard to an explanation of the circumstance that in the different mountain systems of our globe this sickness begins at different altitudes; in the European Alps, for instance, at about 3000 meters (9900 feet) many men suffer, while in the Andes the critical zone is located at about 4000 meters (13,000 feet), and in the Himalayas at about 5000 meters (16,500 feet).

Although the hypothesis that anoxyhemia is the cardinal cause of mountain sickness is at the present time accepted by many authorities, still because there seem to be difficulties in applying this theory to some phenomena, other authors have attempted explanations starting from different points of view.

Kronecker,[5] for instance, assumed that the diminution of the air pressure calls forth a hyperemia and blood stasis of the lungs, which is followed by an affection of the cardiac function and circulation; however, this mechanicophysical hypothesis has no foundation in the findings made by other authors.

Mosso,[8] who, in the Regina Margherita hut on the summit of Monte Rosa, made numerous observations and experiments, gathered experiences that led him to the conviction that the deficiency of CO_2 in the blood, the aknapia, as he names this condition, in contradistinction to asphyxia, was the agent provoking mountain sickness and not the lack of oxygen, the asphyxia of the tissues.

Zuntz,[17] Löwy, Mueller, and Caspari, in their recent work on

the climate of high altitudes, are quite opposed to Mosso's ingenious theory, and the greater weight of evidence substantiates the theory of the anoxyhemia of Jourdanet and Bert.

As it gives the key to a better understanding of the processes of acclimatization, so it likewise enables us to comprehend the differences of the resistance power of the different individuals. In the first place, this depends upon the varying amount of hemoglobin, the oxygen carrier, and upon the form of respiration of the individual. Exact observations and systematic experiments made by Löwy, Zuntz, and others established the fact that deep respirations greatly increase the partial pressure of the oxygen within the lung alveoli, even with unchanged oxygen tension of the outer inspiration air, thus increasing the supply of the blood; simultaneously the mechanical acceleration of the circulation by stronger respiratory movements assures a better oxygen supply of the various tissues.

Therefore, in high altitudes, persons with a type of deep respirations are at a great advantage over persons of flat breathing; this type of respiration being essentially prophylactic against the acute affections of the human organism by a greater decrease of the barometric pressure. On the other hand, a weak nervous system, lack of sleep, disturbances of the functions of the alimentary canal by overeating, alcoholism, etc., increase the liability of the malady and aggravate its character.

Contraindications. Higher altitudes ought to be avoided by persons whose adaptability, on account of certain organic diseases, is below par; for instance, general weakness, or grave degrees of anemia and emphysema pulmonum.

High elevations, even such of 3500 feet, should be considered dangerous, especially for persons suffering from weak heart action, valvular disease, particularly mitral insufficiency and myocarditis, and such pathological changes of the bloodvessels as are found in arteriosclerosis, aneurysma, and nephritis chronica, with high blood pressure.

Most emphatically, in doubtful cases, muscular exercise must be cautioned against until the heart has, to some extent, adapted itself to the changed conditions.

Zangger[16] directs special attention to the dangers of rapid ascent by high mountain railways for elderly persons suffering from nephritis or arteriosclerosis, since syncope, angina pectoris, asthma cardiacum, and apoplexy are possible occurrences also on the return to the lowlands from these excursions.

Prophylaxis and Therapy. In summing up the experiences and the theoretical points, we must accord great prophylactic and therapeutic importance to a sufficiency of the oxygen supply to the organs.

Wherever feasible, some means of transportation (horse, mule, railroad, sedan chair, etc.), should be preferred to exhaustive active

of the symptoms, while vaporous air, cloudy skies, and winds tend to retard or lessen them. A very frequent statement is made that during the night's repose the symptoms become more distressing. Likewise very remarkable is the fact that the symptoms of mountain sickness may appear especially on certain locations of predilection, as is well known on the Alps and the Andes, though other regions of the same mountain range, or of the same mountain even situated higher, may allow mountaineering without suffering. Such places of predilection often are narrow couloirs, ravines, trough-shaped depressions of the niveau, in short, locations more or less protected from the motions of the air, and, therefore, not ventilated. On such places, according to Elster, Geitel, and Zunts, a high ionization, unipolarity, and radio-activity of the air exist as the result of the stagnation of the radio-active emanations from the soil. Saake[10] found the air of high altitudes to possess three times the amount of radio-active emanations than the air of lowlands, and it is known that certain soils and minerals, for instance, those containing pitch-blende or some other metal ores, some clays, etc., emit a greater amount of emanations than others. But further investigation and advance of our knowledge of the influences which the minute electrical and other physical conditions of the atmosphere exert upon the human body, as yet hypothetical, will be necessary before we can pass on the problems that the above-mentioned facts involve. Obscurity prevails also in regard to an explanation of the circumstance that in the different mountain systems of our globe this sickness begins at different altitudes; in the European Alps, for instance, at about 3000 meters (9900 feet) many men suffer, while in the Andes the critical zone is located at about 4000 meters (13,000 feet), and in the Himalayas at about 5000 meters (16,500 feet).

Although the hypothesis that anoxyhemia is the cardinal cause of mountain sickness is at the present time accepted by many authorities, still because there seem to be difficulties in applying this theory to some phenomena, other authors have attempted explanations starting from different points of view.

Kronecker,[5] for instance, assumed that the diminution of the air pressure calls forth a hyperemia and blood stasis of the lungs, which is followed by an affection of the cardiac function and circulation; however, this mechanicophysical hypothesis has no foundation in the findings made by other authors.

Mosso,[8] who, in the Regina Margherita hut on the summit of Monte Rosa, made numerous observations and experiments, gathered experiences that led him to the conviction that the deficiency of CO_2 in the blood, the aknapia, as he names this condition, in contradistinction to asphyxia, was the agent provoking mountain sickness and not the lack of oxygen, the asphyxia of the tissues.

Zuntz,[17] Löwy, Mueller, and Caspari, in their recent work on

the climate of high altitudes, are quite opposed to Mosso's ingenious theory, and the greater weight of evidence substantiates the theory of the anoxyhemia of Jourdanet and Bert.

As it gives the key to a better understanding of the processes of acclimatization, so it likewise enables us to comprehend the differences of the resistance power of the different individuals. In the first place, this depends upon the varying amount of hemoglobin, the oxygen carrier, and upon the form of respiration of the individual. Exact observations and systematic experiments made by Löwy, Zuntz, and others established the fact that deep respirations greatly increase the partial pressure of the oxygen within the lung alveoli, even with unchanged oxygen tension of the outer inspiration air, thus increasing the supply of the blood; simultaneously the mechanical acceleration of the circulation by stronger respiratory movements assures a better oxygen supply of the various tissues.

Therefore, in high altitudes, persons with a type of deep respirations are at a great advantage over persons of flat breathing; this type of respiration being essentially prophylactic against the acute affections of the human organism by a greater decrease of the barometric pressure. On the other hand, a weak nervous system, lack of sleep, disturbances of the functions of the alimentary canal by overeating, alcoholism, etc., increase the liability of the malady and aggravate its character.

Contraindications. Higher altitudes ought to be avoided by persons whose adaptability, on account of certain organic diseases, is below par; for instance, general weakness, or grave degrees of anemia and emphysema pulmonum.

High elevations, even such of 3500 feet, should be considered dangerous, especially for persons suffering from weak heart action, valvular disease, particularly mitral insufficiency and myocarditis, and such pathological changes of the bloodvessels as are found in arteriosclerosis, aneurysma, and nephritis chronica, with high blood pressure.

Most emphatically, in doubtful cases, muscular exercise must be cautioned against until the heart has, to some extent, adapted itself to the changed conditions.

Zangger[16] directs special attention to the dangers of rapid ascent by high mountain railways for elderly persons suffering from nephritis or arteriosclerosis, since syncope, angina pectoris, asthma cardiacum, and apoplexy are possible occurrences also on the return to the lowlands from these excursions.

Prophylaxis and Therapy. In summing up the experiences and the theoretical points, we must accord great prophylactic and therapeutic importance to a sufficiency of the oxygen supply to the organs.

Wherever feasible, some means of transportation (horse, mule, railroad, sedan chair, etc.), should be preferred to exhaustive active

climbing, although the speed of ascent by mountain railroads might for many persons involve an objectionable feature.

Good training of the muscles, heart, and respiratory organs in moderate elevations previous to climbing to higher altitudes is paramount to increasing the resistance power; mounting should be done gradually and leisurely by small stages, so that there may be no abrupt transition, and the climber should be in good physical condition, avoiding extreme fatigue and exertion, which, like sleeplessness, increase the disposition to mountain sickness. The respiration should be deep, the nourishment sufficient and suitable, avoiding digestive disturbances, which by meteorism are apt to interfere with deep respiration.

When travelling by railroads that cross high altitudes, oxygen should be at the disposition of the persons suffering from manifestations, or, as Melinovski, chief engineer of the Oroya Mountain railroad, suggested, cars hermetically closed should be attached to the train, in which a suitable air pressure could be maintained artificially.

When the mountain sickness has set in, absolute rest, deep breathing, or oxygen inhalation will bring temporary relief, otherwise, with more serious symptoms, descent into lower altitudes must be resorted to before a new ascent can be considered.

Against the headache, antipyrine, phenacetine, etc., may be tried.

LITERATURE.

1. Berson und Suering Illustrierte aëronautische Mitteilungen, 1901.
2. Bert, Paul. La pression barométrique, Paris, 1878.
3. Conway. Climbing and Exploration in the Karakoram Himalayas, London, 1884.
4. Humboldt, A. v. Voyage aux régions equinoctiales du nouveau continent, fait en 1799-1804, Paris, 1814.
5. Kronecker. Die Bergkrankheit, Berlin und Wien, 1903.
6. Jaquet, A. Ueber die physiologische Wirkung des Hoehen Klimas, Basel, 1904.
7. Miescher. Correspondenz-blatt f. Schweizer Aerzte, 1893, No. 24.
8. Mosso, Angelo. Life of Man on the High Alps (translated from the Italian), 1898.
9. Mosso, Angelo. La respirazione periodica, Torino, 1905.
10. Saake. München. med. Woch., 1904.
11. Schlagintweit. Result of a Scientific Mission to India and High Asia, Leipzig and London, 1854-58.
12. Schroetter, H. v. Der Sauerstoff in Prophylaxe und Therapie der Luftdruckerkrankungen, Berlin, 1904.
13. Tschudi. Peru, Reiseskizzen aus den Jahren, 1838-42.
14. Whymper. Travels Among the Great Andes of Ecuador, London, 1892.
15. Williams, C. T. Aërotherapeutics, London, 1894.
16. Zangger. On the Dangers of High Altitudes for Patients with Arteriosclerosis, Lancet, 1899.
17. Zuntz and Löwy. Hoehen-Klima und Bergwanderungen, Berlin, 1906.

REVIEWS

MODERN TREATMENT: THE MANAGEMENT OF DISEASE WITH MEDICINAL AND NON-MEDICINAL REMEDIES. By American and English Authorities. Edited by HOBART AMORY HARE, M.D., Professor of Therapeutics and Materia Medica, Jefferson Medical College, Philadelphia; Physician to the Jefferson College Hospital. Vol. I; pp. 930; 105 engravings, and 31 full-page plates. Philadelphia and New York: Lea & Febiger, 1910.

THE notable advance which the past twenty years has brought about in our knowledge of the etiology and management of disease has worked radical alterations in therapeutic procedures. Much, however, of this recent knowledge has been disseminated in a form unavailable for ready application by the practising physician. Therefore, a work such as the one under consideration has become a necessity, and its timely appearance must be regarded with decided satisfaction.

The first part of Vol. I deals with general considerations of drug therapy, and embraces chapters on pharmacology, prescription writing, and the toxic effects of drugs. In the opening chapter, H. C. Wood, Jr., discusses the relationship of modern pharmacology to practical therapeutics. Although he emphasizes the value of pharmacology in medicine, he unfailingly recognizes the limitations of drug therapy. In his excellent summary of the physiological action of drugs, he wisely groups drugs according to their clinical uses rather than into a pharmacological classification.

The chapter on prescription writing, contributed by Hunsberger, is worthy of comment. Written by a skilled practical pharmacist, it is replete with helpful suggestions. Many of the perplexing technical details that frequently confront the physician in his efforts to combine drugs to the best advantage are here explained.

The second part of this volume considers non-medicinal measures for combating disease. It is the largest division of the book, and properly so; embracing nearly four hundred pages, it stands in striking contrast to the hundred odd pages previously devoted to drug therapy. It is impossible, in a review of this length, to take up separately the various important subjects discussed in each chapter. Suffice it to say that in every instance the contributor is one whose ripe personal experience and broad knowledge is such

as to enable him to write with authority upon his particular phase of the subject. The result is a series of admirable essays.

Baruch and Shrady introduce their discussion of hydrotherapy by a timely warning to the medical profession to abandon polypharmacy for the more rational physiological remedies. A consideration of the physiological action of heat and cold precedes their detailed account of the numerous hydrotherapeutic methods and their therapeutic application.

Price has added materially to the value of his article on electrotherapeutics by explaining the physics and physiology of the varieties of electricity, before turning to their diagnostic and therapeutic uses. The careful reading of such a chapter as this would do much to dispel from the minds of many physicians erroneous conceptions, born of insufficient knowledge, of the uses and limitations of electricity in medicine.

The question of nutrition and food has been accorded proper prominence. The subject is well covered by Friedenwald and Ruhräh. After a consideration of the physiology of nutrition and the composition and values of the various classes of foods, they devote several pages to the question of rectal feeding. Infant feeding is taken up in a general way, no effort being made to enter into much detail. On the same broad lines they discuss the dietetic management of fevers, paying special attention to those acute infections in which feeding is of prime importance or of unusual difficulty. Finally, they outline the principles of dietetics involved in various visceral and metabolic diseases, entering more into detail when considering diet in diseases of the gastro-intestinal tract and in diabetes.

Chief interest naturally centres about the chapters on serum therapy and vaccine therapy, since they deal with the most recent evidences of therapeutic progress, and because the results so far obtained in these fields hold out the promise of far greater successes in the future. In these chapters abstruse and confusing subjects are explained in a manner so simple and direct that the reader cannot fail to be enlightened.

Park in his contribution on serum therapy devotes most of his attention to the employment of this form of treatment in diphtheria and tetanus. The use of sera in dysentery, typhoid fever, and cholera, as well as in other acute infections, is, however, given due attention.

The question of opsonins and the opsonic index is taken up by Potter and Avery before they discuss vaccine therapy. Although they admit that the method has been more or less abandoned by many workers, they feel that the last word has not yet been said concerning the true value of the opsonic index. They regard antityphoid vaccination as a procedure of undoubted advantage, and look with decided favor upon the intelligent use of vaccines in the pyococcic infections.

In the comparatively short chapter on glandular therapy, Beebe sets forth the true status of this form of treatment. He divests the subject of all exaggerated claims, at the same time making it evident that the organotherapy of today is based upon the firm foundation of scientific fact.

The last section of the book is devoted to a series of admirable chapters upon the treatment of the most important infectious diseases. The articles in this group, which particularly arrested the attention of the reviewer, were Riesman's chapter on the treatment of typhoid fever, that by Meara on pneumonia and pleurisy, and Landis' consideration of tuberculosis. This last mentioned chapter embodies much sound common sense, and many practical suggestions that cannot fail to be of value to all who are called upon to supervise the management of tuberculosis.

Taken as a whole, Vol. I of this work on treatment is noticeably well balanced, no undue prominence being given any one subject to the exclusion of others. The liberal yet judicious use of illustrations adds decidedly to the clearness of the text in many places. The scarcity of typographical errors is noteworthy in the first edition of a book of this size.

By reason of the wide range of subjects that it covers, and the thoroughly modern, scientific, and, at the same time, practical manner in which these are set forth, it is evident that the volume is destined to assume a leading position among English treatises on therapeutics, and richly merits the unqualified support of the medical profession. G. M. P.

DISLOCATIONS AND JOINT FRACTURES, By FREDERIC J. COTTON, A.M., M.D., First Assistant Surgeon to the Boston City Hospital, Assistant Professor of Clinical Surgery in Tufts College Medical School, Boston. Pp. 654, with 1201 illustrations, 830 from drawings by the Author. Philadelphia and London: W. B. Saunders Company, 1910.

THERE is an immense, indeed an amazing amount of work in this volume, showing that the author has cogitated and digested, argued and proved (or disproved), the matter of which it is composed many a time and oft during the five years or more which have elapsed while the book was making. It is a book entirely unlike most others, and herein consist its merits and its faults. Dr. Cotton says he has made an attempt to throw overboard all hereditary surgical views, to state what is really *known* of the subject today—to make a fresh start, so to speak, in this very important branch of surgery. He states with *naïveté*, "Wisdom did not begin with this generation, but we have had an unusual opportunity to learn,"

largely from the use of the *x*-rays and more frequent operative intervention. He boasts entire and sole responsibility for the contents of the volume; no one else has in any way assisted in its preparation, either as regards text or illustrations; the "first draft" of the work burst fully grown and clothed from his brain, like Minerva from the head of Jupiter; and references to "literature" collected later, were used mostly "for verification of fact and perspective, not for incorporation in the text." He acknowledges that perhaps this is not the right way to work, but excuses it as a reaction from the "benumbing German scholasticism," in which he was trained.

After an Introduction and a chapter on "Generalities," he discusses regionally the dislocations and joint fractures of the various parts of the skeleton, including jaw, spine, thorax, upper extremity, pelvis, and lower extremity.

He has a proper appreciation of the futility of expecting absolutely accurate apposition in fractures of the shafts of long bones, and of the misleading impression given to courts and laity by *x*-rays of end results. Yet he justly insists on exact reduction in fractures near joints, as restoring the contour necessary for useful function, and lessening the amount of callus formed. His views as to earlier (not necessarily *early*) motion of joints, so long as pain is not produced, will be commended by all modern surgeons; but his reference to the "frenzied lead" of some French surgeons (pp. 38, 39) perhaps indicates an imperfect comprehension of what really are the teachings of Lucas-Championnière and his followers. He claims that operation in recent fractures is now too indiscriminate, and he suggests, without expressing it, what is, no doubt, an absolute truth, that surgeons who are most insistent advocates of this treatment are those who are unable to secure by non-operative means as good results as their more conservative colleagues.

Dislocations of the shoulder, he holds, usually are produced not over the acromion as a fulcrum, but by the action of the tense adductor muscles with the arm in extreme abduction. His condemnation of the Velpeau bandage (p. 168) may depend on his ignorance of its proper application; certainly the curious arrangement shown in Fig. 119 is not a Velpeau bandage. He persists, like a few other surgeons, in calling an angular splint applied to the flexor surfaces of the arm and forearm an "internal" angular splint; whereas in exact parlance this term should refer to the Physick splint, and such a splint as Hartshorne's is more accurately designated an "anterior" angular splint.

In discussing lesions of the elbow Dr. Cotton is somewhat contradictory in stating (p. 212) that children are apt to fall on the elbow itself, not on the outstretched hand, thus explaining the less serious injuries encountered in childhood; while at p. 243 he states that the severe injuries met with in adults are to be explained on the score of direct violence, adults falling on the elbow, or having

it crushed in machinery, etc. It is also categorically stated (p. 273) that "separation of the *whole* lower epiphysis is possible up to four years of age," while Fig. 355, of a "boy of twelve years" is described as a "separation of the whole lower epiphysis of the humerus." In two foot-notes he attacks Dr. Warbasse for ignorance; and yet on the latter page exhibits his own by the bold assertion that in fractures of the external condyle "lateral mobility of the forearm as a whole does *not* occur;" and at p. 290 by his denial that physiological *cubitus valgus* owes its existence in part to a normal obliquity in the joint surface of the bones of the forearm at the elbow as well as in that of the humerus. In the treatment of supracondylar fractures at the elbow he is partial to a right angled moulded tin splint; but it can scarcely be doubted that if he reduced these fractures completely and put them up in hyperflexion (not merely acute flexion, as shown in Fig. 393), his results would be better, especially as regards the occurrence of *cubitus varus*, than those he has recorded elsewhere.

The merits of the book are many; its faults are not a few. It is suggestive, but it will serve no useful teaching purpose unless revised. It is weak in pathogenesis. There is endless tabulation of varieties and subvarieties of lesions, but there is no real classification. Rare lesions are often seen through a magnifying glass, but the myopia is too great to allow a vision in perspective. The illustrations are excellent (save a few which appear as if seen through a *camera obscura*, and a fewer still which look like nightmares), but they would have been still more excellent if drawn or even retouched by a real artist. The references to "literature" are fairly numerous, but valuable contributions (*e. g.*, Quénu and Küss on luxations of the metatarsals, the type of elbow fracture associated with the name of Prof. Posadas, etc.) would not have been overlooked if the author had had less contempt for scholasticism; and if he had enlisted friendly advice and criticism in the preparation of the text, so many contradictions and inconsequences would not have passed unobserved.

The work is in no sense a text-book. Its perusal, were such a thing possible for an undergraduate student, would give him such a confused and kaleidoscopic idea of dislocations and joint fractures as to unsettle his surgical reason forever. For those who have already learned a certain amount of perspective, and who can catch glimpses of the distant landscape through mists of beclouding detail, the volume is a storehouse of information, ideas, helps, suggestions, and inspirations to revise, correct, and improve their own practice.

A. P. C. A.

OPERATIVE SURGERY. By JOHN J. MCGRATH, Professor of Operative Surgery at the New York Post-Graduate Medical School. Third Revised edition; pp. 653; 276 illustrations, including many full-page plates in color and half-tone. Philadelphia: F. A. Davis Company.

IT is stated in the preface of this book that particular care has been given to the section on abdominal surgery, and as a review of previous editions has been published, it may be well to discuss this section alone. The anatomy of the walls of the abdomen is well described, and the various methods for its incision are recounted. In the description of the operation, one might point out: That it is not necessary to pocket the subaponeurotic space in performing the Mayo operation for umbilical hernia; that Moynihan's method for total gastrectomy is somewhat better than the one described; that enterostomy for typhoid perforation as prescribed by Hays is omitted; that no mention is made of the "step" commonly practised in performing posterior gastrojejunostomy, whereby the edges of the transverse mesocolon are fastened either to the stomach or to the line of suture; that it is totally unnecessary and dangerous to dilate the stump of the appendix in order to invert it.

There is no mention of any of the operations for tumors of the bladder nor of the osteoplastic flap method in performing laminectomy. Silk or even silkworm gut is advised for suturing in the repair of hernia.

It is difficult to criticise a book of this character, because it consists, practically, of a compilation of established methods of surgical technique, but it does seem as though in a future edition the author should bring many of the operations more strictly "up to date." The greater part is excellent, and the volume of a handy size, well printed, and having comparatively few but generally adequate illustrations. G. P. M.

BISMUTH PASTE IN CHRONIC SUPPURATIONS. By EMIL G. BECK, Surgeon to the North Chicago Hospital, Chicago, Illinois. Pp. 237; 81 engravings, 9 diagrammatic illustrations, and a colored plate. St. Louis: C. V. Mosby Co., 1910.

SINCE Beck, in 1906, introduced the use of bismuth paste in the treatment of chronic suppuration there has appeared a great deal of literature upon the subject. This book, by the originator of the method, is especially timely as expressing the collective experience of the one most qualified to write on the subject. The contents include the use of the paste in diagnosis, in the treatment of sinuses and fistulæ, its limitation and the causes of failure, bismuth poisoning and its prevention, and its use in nose and ear work and

in dentistry. The text is lucid and exceedingly interesting—almost wonderful—the illustrations particularly good, and the size of the volume shows excellent judgment, there being no padding whatever. The method has made easy the treatment of conditions heretofore the bane of surgical practice, and nowhere can a clearer understanding of the subject be attained than in this book.

G. P. M.

DIE WASSERMANNSCHE REAKTION MIT BESONDERE BERÜCKSICHTIGUNG IHRER KLINISCHEN VERWERTBARKEIT. Von PRIVAT-DOZENT DR. HAROLD BOAS, I Assistenten am Rudolf Berghs-Hospital für venerische Krankheiten in Kopenhagen, mit einen Vorwort von GEN. RAT. PROF. DR. A. WASSERMANN. Berlin: S. Karger, 1911.

THIS book of something over 180 pages is founded principally upon the personal observations of the author, who has had a very large and interesting experience with the Wassermann reaction. His conclusions, therefore, bear the mark of individual work, and are for this reason of special value. The entire subject is discussed in full and with a wide knowledge of the literature, a task which today has become most difficult, since the bibliography is truly appalling, covering in this treatise 44 closely written pages.

After an historical introduction, the technique is discussed in the second chapter. It is to be noted here that the Noguchi method is most inadequately described. Chapters follow upon control cases and the Wassermann reaction in the various stages and types of syphilis, and upon the influence of antisyphilitic treatment upon the reaction. Finally the question as to the meaning of a positive Wassermann reaction is discussed, and in this chapter the author upholds the view that a positive reaction indicates active syphilis.

W. T. L.

DIFFICULT LABOR. A GUIDE TO ITS MANAGEMENT. By G. ERNEST HERMAN, M.B., F.R.C.P., Consulting Obstetric Physician to the London Hospital. Fifth edition; pp. 547; 180 illustrations. New York: William Wood & Co., 1910.

As this book has now reached its fifth edition, any review in the true meaning of the term would be superfluous, and the duty of the reviewer is simply to call attention to the more important changes and additions contained in the book of 1910, as compared with its predecessor of 1896. The chief additions have been chapters upon retroversion of the pregnant uterus and upon puerperal eclampsia.

The apology made for their introduction upon the ground that they are not complications of labor is unnecessary, since both at times do complicate the child-bearing process, and, in any event, are closely related to it. The first of these two chapters may be dismissed with the statement that it is in keeping with the remainder of the work, than which no criticism could be more favorable; but this cannot be said with respect to the chapter devoted to eclampsia. This portion of the book is hardly abreast with present-day teaching, and a careful reading will leave one with a suspicion that the author did not desire that it should be. Aside from its last paragraph, consisting of instructions as to treatment by morphine and hydrotherapy, it is somewhat too vague to be of much practical value, both as regards pathology and treatment. While it is universally admitted that our ignorance as to the essential etiology of eclampsia is abysmal, it would certainly have served a better purpose to have considered the subject from the basis of the theories of auto-intoxication rather than to have made a thoroughly artificial division, admittedly based upon an imaginary pathology. The most glaring defect in the chapter, however, is the hopeless attitude with regard to prophylaxis. This is pernicious teaching in the light of common present-day knowledge. It is to be hoped that in the editions of the future the author will see fit to bring this chapter up to the standard of excellence of the remainder of the work. W. R. N.

PRACTICAL OBSTETRICS. By E. HASTINGS TWEEDY, F.R.C.P.I., Master of the Rotunda Hospital, Dublin; and G. T. WRENCH, M.D., Late Assistant Master. Second edition; pp. 491; 159 illustrations. London: Henry Frowde, and Hodder & Stoughton, 1910.

THIS book, as is shown by its title, aims to present simply the practical side of obstetrics. It is really a rather elaborate compend or syllabus, as those terms are understood in this country; in other words, it is a book which will be useful to the student preparing for examination or to the ill-trained practitioner who desires to become conversant with the elements of obstetrics. It could not be used as a text-book in American medical schools of the first class, as it comprises simply the practice of the Rotunda Hospital and does not pretend to give an exhaustive survey of either the theoretical or practical aspects of the subject. Its review has been of decided interest, since it is an authoritative expression of the technique of a very famous institution. Of course, it goes without saying that in the main its teaching is the result of great experience and that it is eminently sound, but there are a few exceptions. For instance, we have been greatly interested in the advised avoidance of both bleeding and sweating in the treatment of eclampsia; in the statement

that patience is the only treatment of an undilating os, the result of primary inertia; and the implied advocacy of quinine, ergotinin, and treacle as stimulants in uterine inertia. While by far the greater number of subjects are well and clearly handled, the description of vaginal Cesarean section and uterine suspension are not given in sufficient detail to allow of their comprehension by the ordinary reader. The advocacy of complete anesthesia for forceps delivery is certainly dangerous teaching if followed by the general practitioner in his every-day practice. Those who follow it conscientiously will undoubtedly gain a rather wide experience in postpartum bleeding. It seems rather foolish to advocate the use of the Skutsch pelvimeter to men whose work is of necessity done in private houses and in many instances among the poor. This instrument is undoubtedly of value in trained hands, particularly in hospitals where ample assistance is available, but the ordinary practitioner would certainly obtain naught but misleading evidence from its use. The usual method of estimation by means of the diagonal conjugate as measured by the examining finger is amply sufficient for his needs. In the performance of abdominal Cesarean section it hardly seems needful to compress the broad ligaments during the incision of the uterus or to dilate the cervix afterward, even if there were no dilatation. Certainly the use of the finger as the dilating force would not appeal to many obstetricians.

While, as has been already stated, the book fulfils its purpose quite well, it is nevertheless somewhat of a disappointment, since a different treatment of "practical obstetrics" might be expected from men of the prominence and experience of the authors of the present volume. W. R. N.

MEDICAL ELECTRICITY AND RÖNTGEN RAYS, WITH CHAPTERS ON PHOTOTHERAPY AND RADIUM. By SINCLAIR TOUSEY, A.M., M.D., Consulting Surgeon to St. Bartholomew's Clinic, New York. Pp. 1116; 750 illustrations. Philadelphia and London: W. B. Saunders Company, 1910.

LIKE other books written upon electricity, it first deals with general considerations and principles, and then takes up the special currents.

The illustrations are good. The book is well gotten up, and it is indeed a welcome sight to see new plates for motor points and not the old Erb plates, which are used in practically every text-book.

It is difficult to review a large work of this kind, but the first impression is that the book is rather too large. Not that comprehensive treatment is out of place, but there is a lot of repetition of theories by other authors, not only of general principles, but of therapy, and the consequence is that many statements are made of cures of diseases by different forms of electrical application which are

not borne out by facts, but which the author quotes in good faith from men who apparently should know. The truth of the matter is, that no one person is qualified to write upon all the different phases of electricity and its treatment. A man who devotes his time to *x*-ray work cannot very well be expert in the general treatment of nervous diseases by electricity, and the same thing holds true for the neurologist.

The reviewer is a neurologist, and therefore can only review the book from his standpoint. For example, in the treatment of sclerodema the statement is made that "perforating ulcer of the foot has been cured by faradization of the posterior tibial nerve and erythromelalgia and symmetrical gangrene have been successfully treated by the local application of galvanic currents." This is a rather strong statement, and one which the reviewer wishes were true. Again, the statement is made that a "galvanic hydroelectric bath for sciatica cures the majority of the cases." Every neurologist is at times compelled to use electricity in the treatment of neuritis, but unfortunately not always with good results. It is difficult to understand why manufacturers of electrical apparatus always persist in having their electrodes covered by sponges when a metal disk with cotton is so much better and cleaner, and yet in most of the illustrations sponge electrodes are shown.

The chapter on nervous diseases is particularly well done, and the description of the various palsies, disturbance of sensation, etc., is up to date, and is to be commended. One is interested in seeing a description of Laduc's treatment of trigeminal neuralgia by means of cataphoresis, and the author is correct in rather doubting its efficacy.

It is rather difficult to understand the following. In discussing the value of high-frequency currents, the author states that he has had some success in the treatment of infantile paralysis, and then he mentions the following extraordinary case on page 542: "One poor little hand was crippled and about one-half the natural size, and the leg on that side was in about the same condition. A few months' treatment with a vacuum electrode connected directly with one pole of an *x*-ray coil brought the paralyzed arm and leg up to a size exceeding those of the normal limbs originally, and at the age of two and one-half years the boy weighed forty pounds, and was strong and jolly." He further recommends high-frequency treatment for epilepsy, chorea, and locomotor ataxia, stating that in the latter disease such marked improvement resulted in a case that "the patient's friends have stopped him on the street and asked him what treatment he was receiving." Such statements do not sound well.

Since the introduction of the *x*-rays in 1895 the advances made in it are remarkable, and the author discusses this part very well. The plates are excellent. On the whole, the book is good, and the author should be commended for his work.

T. H. W.

PROGRESS

OF

MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

REGIUS PROFESSOR OF MEDICINE, OXFORD UNIVERSITY, ENGLAND,

W. S. THAYER, M.D.,

PROFESSOR OF CLINICAL MEDICINE, JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND,

AND

ROGER S. MORRIS, M.D.,

ASSOCIATE IN MEDICINE, JOHNS HOPKINS HOSPITAL, BALTIMORE, MARYLAND.

A Sulphur Reaction in the Urine in Cancer.—H. SOLOMON and P. SAXL (*Wiener klin. Woch.*, 1911, xxiv, 449) have made a further study of the oxyproteinic acid of the urine in carcinoma. It is fairly rich in incompletely oxidized sulphur, and Solomon and Saxl have devised a method for oxidizing the sulphur and determining the approximate quantity of sulphate resulting. The method is as follows: 150 c.c. of urine are diluted with 100 c.c. of distilled water. (Albuminous urines must first be boiled, acidified with dilute acetic acid, and filtered.) One then adds 150 c.c. of baryta mixture to remove the sulphates (Salkowski). Baryta mixture consists of two parts of a solution of barium hydroxide saturated at room temperature and one of barium chloride, also saturated at room temperature. (The salts must be chemically pure and kept in stoppered bottles to prevent efflorescence. The saturated solutions are filtered clear.) After adding the baryta mixture, filter and test a small portion of the filtrate with a few drops of baryta to see whether precipitation has been complete. Then, to remove the ethereal sulphates (according to Salkowski), 300 c.c. of the filtrate are taken, to which 30 c.c. of HCl (sp. gr. 1.12) are added, and the whole placed in a 500 c.c. Erlenmeyer flask. The latter is covered with a small funnel, placed on asbestos and boiled one hour over a small flame. (One must guard against change in the HCl concentration from this point onward, hence the use of the funnel in the mouth of the flask.) After boiling, the flask is covered with

a beaker and placed on the water bath, where it remains till the fluid overlying the precipitate is clear, usually four to twenty-four hours. Now one filters twice through a double, dry baryta filter (Baryt-filter)—use a small filter—washes the Erlenmeyer flask with alkali, water, and distilled water, and then boils again for a short time in the same flask with the funnel in place. Again one filters through a double baryta filter. (If a precipitate remains on the filter, the flask is returned to the water bath.) Two hundred c.c. of the filtrate are now treated with 3 c.c. of Merck's perhydrol (hydrogen dioxide) and boiled one-quarter of an hour in the same Erlenmeyer flask with the funnel in the neck. After boiling, the fluid is poured into a conical sedimenting glass, and within one-half to four hours one sees a precipitate settling to the bottom. This precipitate is abundant only in cancer. Ordinarily, in normal urines there is no precipitate, or at most a trace within four hours. Usually, much later a precipitate appears, which is possibly barium sulphate derived from the ethereal sulphates. The precipitate in cancer is barium sulphate plus pigment; the latter is removable by extraction in alcohol-ether. What remains is sulphate obtained by oxidation from the neutral sulphur. Solomon and Saxl have tested the urine from 81 cases of cancer, of which 61 gave a markedly positive reaction, 10 weakly positive, and 10 negative. A negative result was obtained in 79 controls. Three cases which were positive remained clinically doubtful, being apparently myoma uteri, pseudoleukemia, and anemia gravis. The reaction may appear early, even with very small tumors, and appears to be practically specific.

Diagnostic Value of the Butyric Acid Test (Noguchi) in Cerebrospinal Fluid.—In the routine study of all spinal fluids sent to the laboratory of the Michael Reese Hospital for diagnosis, STROUSE (*Jour. Amer. Med. Assoc.*, 1911, lvi, 1171) has studied particularly the status of the butyric acid globulin test as devised by Noguchi. In 5 cases of cerebrospinal syphilis, and in 7 of general paresis, he found the Noguchi test positive. In 2 cases of tabes the test was positive, and in 2 negative. The Wassermann test in the blood of one of the latter was positive. In contrast to this series are 3 cases of brain tumor, 2 of dementia præcox, and 2 of cerebral arteriosclerosis, in all of which the Noguchi test was negative. As an aid in the differential diagnosis between acute meningitis and other inflammations of the meninges, Strouse found the test positive in 4 cases, 1 each of pneumococcus, meningococcus, streptococcus, and influenzal meningitis. In contrast is a negative test in 3 cases of meningismus, 2 in pneumonia, and 1 in typhoid fever. He found positive Noguchi reactions in 18 cases of tuberculous meningitis, all of which later gave a positive diagnosis by the finding of tubercle bacilli or by autopsy. The test was more constant than lymphocytosis and was much easier to perform than a total or differential cell count. In 9 cases in which the tentative diagnosis of tuberculous meningitis had been made, the test was negative and the later course proved the diagnosis to be incorrect. Warning is given, however, that even in clinical conditions which may resemble tuberculous meningitis a positive reaction does not by any means indicate the presence of a tuberculous inflam-

mation. A negative one presupposes the absence of such a condition. He concludes that the tests for increased globulin in the spinal fluid are easier to perform than the total cell count, and have practically the same diagnostic importance. The butyric acid test of Noguchi is convenient, accurate and gives information of considerable value in diagnosis. He employed the test as follows: One part (0.1 or 0.2 c.c.) of cerebrospinal fluid is placed in a test-tube and to this is added 5 parts of a 10 per cent. solution of pure butyric acid in physiological salt solution; the mixture is heated to boiling and immediately one part of normal sodium hydrate added and the mixture again boiled. In the presence of increased globulin a definite flocculent precipitate occurs, either immediately or within two hours. This definite precipitate must be differentiated from a faint cloudiness which normal fluids may give, but it requires only slight experience to recognize the difference between positive and negative reactions.

Peptid-splitting Ferment in the Saliva.—The glycyltryptophan test for the diagnosis of gastric carcinoma, as described by Neubauer and Fischer and the results obtained by Lyle and Kober, and by Weinstein, have been reported in previous numbers of this journal. A striking feature of their reports has been the consistency with which the reaction has stood the test of application. In the presence of anacidity and the absence of pancreatic juice and blood, the stomach juice which has split the polypeptid glycyltryptophan has been said to contain a proteolytic enzyme derived from carcinoma. WARFIELD (*Johns Hopkins Hosp. Bull.*, 1911, xxii, 150), dissatisfied with discordant results, investigated the effect of saliva upon glycyltryptophan and was surprised to find the presence of an enzyme which split tryptophan from the dipeptid. This enzyme is destroyed by heating to 100° C. and is not active in acid saliva. Warfield explains the presence of the test in achylia gastrica by the presence in an alkaline medium of swallowed saliva. An occasional acid saliva would explain an occasional negative test. When the stomach contents are acid, 0.05+ per cent. the test is negative. Warfield acknowledges the presence of a peptid splitting enzyme in the cancer juice, but points out that the absence of free hydrochloric acid in the stomach contents in cases of carcinoma presents a condition most favorable for the action of the salivary peptid splitting enzyme. He has not determined from what glands, parotid or submaxillary and sublingual, this enzyme was secreted. He has not determined the effect of the bacteria in the saliva. In view of these facts, he concludes that the glycyltryptophan test is of no value in the diagnosis of cancer of the stomach.

Cardiac Insufficiency, Albuminuria, and Cylindruria Caused by Chronic Coprostasis.—EBSTEIN (*Münch. med. Woch.*, 1911, lviii, 615) reports his clinical findings in several recent cases. His observations have led him to believe that when the cardiac resistance as determined by tactile percussion is widened, one possibility to be considered is dislocation of the heart from a high position of the diaphragm. If such be the case, the cardiac symptoms, which may amount to a muscular insufficiency, may be cured if it is possible to cause the

diaphragm to return to the normal level. Chronic coprostasis is a frequent cause of elevation of the diaphragm. Again, before giving an unfavorable prognosis because of albuminuria and cylindruria, one must relieve a chronic constipation, since both may disappear with the establishment of normal fecal evacuations.

A New View of the Function of the Renal Glomerulus.—Brodie (*Jour. Can. Med. Assoc.*, 1911, i, 18) discusses a new view of the function of the renal glomerulus. On the assumption that the glomerulus discharges the bulk of the water and does actively secrete, he says that, since the abandonment of the filtration hypothesis, there has been no explanation of the function of this characteristic and peculiar structure. He feels that the glomerulus actively sets up a pressure head whereby the column of fluid is propelled along the tortuous tubule; for it seems reasonable to suppose that a considerable head is necessary to force fluid through the tubule, as it is a capillary tube. He observed the rate of flow from a dog's kidney in active diuresis; ligated the vessels; fixed the organ in formalin, and found out the number of glomeruli in each kidney, thus getting an idea of the rate of flow from each tubule. From the law of motion of fluid in a tube of that diameter he found the average glomerular pressure to be about 95 mm. Hg. All attempts to show that any renal cells can give an excretory pressure similar to that of the salivary glands failed, and consequently we must assume that the pressure just outside the glomerular tuft is the same as the renal capillary pressure. This renal capillary pressure is about 30 mm. Hg., less than arterial pressure, that is, 95, the same as found necessary to propel the fluid through the tubule. The popular view explains many facts brought to the support of that of filtration, that is, why the tuft has the firm unelastic Bowman's capsule and why the afferent vessels are larger than the efferent. This arrangement permits of a rapid enlargement of the glomerulus without loss of pressure. Nussbaum has described a pulsation of the glomerulus in the newt, and the tubules in active diuresis have been found increased in length. Both of these conditions are due to increased glomerular pressure. Furthermore, it has been found that the urinary secretion ceases at a pressure of about 30 to 40 mm. Hg, lower than the arterial pressure, that is, when the urinary pressure equals that in the glomerulus. Caffeine diuresis has been ascribed to the vessles alone, but proof is now at hand that caffeine products exercise a marked influence on the renal cells, and that under their influence the cells become incapable of holding back chlorine from the blood, after the chlorine content has fallen below normal. Then experiments on the frog's kidney where glomeruli and tubules have a distinct circulation leave no doubt that under caffeine diuresis, the tubules secrete, hence, in nephritis, great care should be taken in caffeine administration.

The Sensibility of the Alimentary Canal in Health and Disease.—Hertz (*Lancet*, 1911, clxxx, 1051), in the Goulstonian lectures, records his observations on the sensibility of the gastro-intestinal tract to various stimuli. The mucous membrane of the whole canal was found insensitive to tactile stimuli; that of the stomach and intestines alone was insensitive to thermal, the esophagus and rectum being sensitive.

The esophagus and stomach are quite insensible to dilute hydrochloric acid even as high as 5 per cent., a concentration never found in health or disease. Glycerin affects the anal canal, and alcohol generally produces a feeling of warmth. The feeling of fulness in stomach and intestines is due to a slow increase in the tension exerted on the muscular fibers of their coat. The volume of contents necessary to produce this tension varies with the tone of the organ, that is, a small quantity will cause a sensation of fulness in a tonically or organically contracted stomach, while it would be unnoticed in the dilated. In the rectum this special sensation of fulness is a "call to defecation." This call should be answered at once; for the passing off of the desire is not due to a return of the feces to the pelvic colon, but to a gradual relaxation of the rectal wall. A new desire means only one thing—that more feces have come to the rectum, and to prevent progressive dilatation it should be evacuated at once. The feeling of hunger is one of general malaise associated with the periodical muscular activity of the stomach when empty and the nerves hypersensitive. This causes local pressure symptoms analagous to those to be described in pain. Hunger can be allayed by the taking of water or of any non-digestible substance like earth. It is, however, with especial reference to abdominal pain that his observations are most interesting. He has described how he found the stomach insensitive to hydrochloric acid, even when ulcerated, and believes that abdominal pain is, in part, truly visceral and has one cause —tension produced upon the muscular coat of the viscus. This tension must be induced rapidly; if slowly the sensation of fulness results, and the pain returns when more pressure is added. Gastric pain associated with ulcer he describes as due to the pressure caused by (1) pyloric spasm either organic or due to the passage of too acid chyme into the duodenum, together with (2) increased peristalsis of the pyloric end of the stomach. This rapidly raises pressure locally, and the pain is severe. The peristalsis may be more active from direct stimulation of the nerves of the open ulcer, or it may be due to plain hyperacidity. If the ulcer is in the cardia, the peristalsis is evidently greatly increased, reaching a region of the stomach where it normally does not belong. The late pain of duodenal ulceration he explains by the fact that the first chyme to pass the pylorus has hydrochloric acid bound with the protein, and that there is no excess to cause pyloric spasm. As the secretion of hydrochloric acid rises, the spasm increases and peristalsis causes the rise in pressure. Relief by soda bicarbonate is explained by the reduction of acidity and consequently of the pyloric spasm, thus permitting extrusion of chyme. Similarly, vomiting results in emptying of the stomach with lack of pressure owing to their being nothing for the stomach to contract upon. The pain in chronic appendicitis and cholilithiasis is most probably due to reflex inhibition of the pyloric relaxation. That of colic is due to increase of pressure caused by excessive peristalsis and spasm of the next lowest colonic segment. Local tenderness may be due to two things—hyperalgesia of the skin, subcutaneous tissues, and muscles, together with a local rise in tension, caused by the pressure of the hand. That it is not due to pressure on the ulcer is evident from the fact that exploratory laparotomies often show the ulcer to be remote from the tender spot.

The Relationship of Electrocardiographic Methods to Medicine.—LEWIS (*Proc. Roy. Soc. of Med.*, 1911, iv, 81, medical section) discusses the relationship of electrocardiographic methods to medicine. Its principal work has been to clear up the mystery surrounding delirium cordis and paroxysmal tachycardia. The delirium cordis is definitely due to the experimentalists' fibrillation of the auricles, and this is accountable for 50 per cent. of all irregularities; it is of further importance because it reacts to drugs in a specific manner. Cases of paroxysmal tachycardia may be placed in several classes. The first show crises of auricular fibrillation. In the second the paroxysms depend upon new impulse formation in the myocardium at a point remote from the normal impulse formation area; the attack depends upon the substitution of a new rhythm for the old. With this discovery has come the localization of the pacemaker in the right auricle just at the entrance of the superior vena cava. While the instrument lacks the portability of the polygraph, it can be carried remotely by wires, and it gives quickly an infallible analysis of the sequels of contraction in the cardiac chambers (not of the effects of contraction on the veins, as does the polygraph) without any measurement. Finally it gives information of the direction pursued by the contraction waves. It must be given a place with percussion and auscultation. Its information is direct and definite whether given or withheld.

The Behavior of Various Organic Acids Concerned in Diabetic Acidosis.—LABBE and TROLLE (*Presse Méd.*, 1911, p. 284) have investigated upon rabbits the various organic acids supposed to be concerned in diabetic acidosis. After showing that mineral acids either killed quickly by intense action or could not produce coma, they tried propionic, lactic, butyric, and beta-oxybutyric acids successfully producing coma with all. Independent of the toxicity due to the acid, is a specific toxicity due to the chemical constitution, and those without the alcohol radicle are the more dangerous. One can diminish the first toxicity due to the acid by saturation of the animal with alkali, but the specific toxicity is not affected; for example, if it takes 375 grains of soda butyrate to kill an animal, the corresponding dose of acid is 90 grains. As this acid content can be neutralized, they believe in the free administration of alkalies, carbonate of soda preferably, and also in the exhibition of alcohol, since those with alcohol in the radical are the less toxic. Most failures are due to a failure of saturation by the alkali treatment.

Urobilinogenemia.—R. VON JAKSCH (*Münch. med. Woch.*, 1911, lviii, 746), in referring to the work of Hildebrandt (*Ibid.*, 1910, lvii, 2574), who claims to have been the first to demonstrate urobilinogen and urobilin in the blood serum, shows that the latter is mistaken and points to an earlier reference. Von Jaksch also states that for years he has observed urobilin in the blood serum in the lobar pneumonia. In his experience, urobilinogenemia has always been associated with severe, fatal infections. No hepatatitis has been found associated at autopsy. The serum from such a case, at first yellow, gradually assumes a greenish fluorescence on prolonged exposure to the light. No bilirubin has been demonstrable in the serum.

SURGERY

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

FORMERLY JOHN RHEA BARTON PROFESSOR OF SURGERY IN THE UNIVERSITY OF PENNSYLVANIA, AND SURGEON TO THE UNIVERSITY HOSPITAL,

AND

T. TURNER THOMAS, M.D.,

ASSOCIATE IN SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE PHILADELPHIA GENERAL HOSPITAL, AND ASSISTANT SURGEON TO THE UNIVERSITY HOSPITAL.

Jejunal Ulcer after Gastro-enterostomy.—EXALTO (*Mitth. a. d. Grenzgeb. d. Med. u. Chir.*, 1911, xxiii, 13) says that since Braun, in 1899, demonstrated the occurrence of ulcer of the small intestine following a posterior gastro-enterostomy, only a relatively small number of cases has been reported. Probably many more occur, but because of favorable circumstances are healed. The known cases may be divided into those in which perforation occurs into the free peritoneal cavity, and those in which the ulcer adheres to the anterior or posterior abdominal wall and is thus encapsulated. All writers have discussed the diagnosis and therapy and especially the obscure etiology. Almost all have based their conclusions upon speculation without experimental evidence. Exalto performed a series of experiments on dogs with the object of studying the effect of the gastric juices on the jejunal mucous membrane. He concluded that the ulcer following gastro-enterostomy is the result of the effect of the acid gastric contents upon the intestinal mucosa. It remains undecided whether the lesions are due to mechanical trauma or other causes primarily present; or whether the chemical trauma is of too high a grade for the life of the epithelial cells of the mucous membrane. The Roux Y method of operation on the anterior or posterior wall, and those methods which are associated with a Braun anastomosis (entero-anastomosis), must be avoided. The method which affords the least possibility for the development of a jejunal ulcer, is the retrocolic posterior gastro-enterostomy (v. Hacker), as near as possible to the duodenum. The after-treatment for persons upon whom the operation has been performed for ulcer should provide for an ulcer diet for a long period, with the administration of alkaline substances, since this is an important factor in the prevention of jejunal ulcer.

The Anatomical Basis for Surgery of the Lymph Nodes; The Regeneration and New Formation of the Same.—VECCHI (*Mitth. a. d. Grenzgeb. d. Med. u. Chir.*, 1911, xxiii, 42) says that conclusions based upon clinical observations have little support. Only experimental evidence has any value. Vecchi carried out a series of experiments upon dogs, and as a result concluded that after a total enucleation of a lymph node, no regeneration occurs. The symptoms which develop as a consequence of the operation (lymph effusion, scar formation,

edema, etc.) after varying periods of time disappear. The lymph circulation is carried on partly through preformed or new-formed collateral vessels, and in part through the old path which is in most part reëstablished. After a resection of a lymph node, regeneration of the resected portion never takes place. Healing occurs by the development of a scar. The behavior of the remaining portion of the node will depend upon the direction and extent of the resection. By the injection of certain substances into a lymph node, an experimental exuberance can be produced, such as occurs in certain pathological conditions (inflammations, malignant tumors). Under the influence of the long-continued irritation, the lymph follicles grow toward the periphery and form plugs and buds which project into the perinodular connective tissue. They may become detached from the node and form daughter nodes. These occur especially in the direction of the lymph stream, along the afferent vessels of the wounded lymph node. It is not an autogeneration but an outgrowth. By the formation of a connective-tissue septum in an altered node, the latter may be divided into two portions, which may be mistaken for newly formed nodes.

Suction Treatment in Sterile Sand Baths of Infected Wounds and Wounds Suspected of Being Infected.—Thiers (*Zentralbl. f. Chir.*, 1911, xxxviii, 458) has employed in many severely infected wounds, with much satisfaction, a method of treatment which is based upon the ability of fine sand to exert capillary attraction upon fluids. The finer the grains of sand the greater the suction. The suppurating wound or sinus is covered with sand until the involved portion of the body, as the forearm, lies in the sand bath. A quadrilateral tin box or gutter, with removable or very low ends, answers the purpose. The secretion is quickly drawn from the wound, so that the sand in the immediate neighborhood of the wound should be changed frequently. This the patient can manage with a spoon. To sterilize the sand it should be placed in a linen bag holding about 3 to 4 liters of sand, with the neck of the bag tied; it should be placed in a suitable vessel, filled with a 1 per cent. soda solution, and boiled about a half hour. The alkaline reaction of the sand will prevent the clotting of substances from the wound and the tendency to scabbing. The sand should be sterilized daily or every two or three days. The development of a putrid odor in the sand will call for frequent changing of it. Frequently the application of the sand bath for twelve to fourteen hours will be sufficient. If during the night marked swelling and redness return, a re-immersion in the sand bath will cause them to disappear, frequently in two to three hours. Occasionally the suction is so strong and the discharge so free, that the patient becomes exhausted, and the bath must be discontinued after a short period. In such a case Thiers found it necessary to remove the wound from the bath from time to time and to employ sand with larger grains. For this treatment are found most suitable, large wound surfaces with marked infiltration, wounds with flabby and hypertrophic granulations showing no tendency to cicatrization, and putrid infections with much secretion and showing much edema. The edema and inflammation quickly disappear, the granulations become full and bright red, and the cicatrization progresses favorably. The pain also disappears in a similar manner and the

mobility of joints is improved. The sand bath is particularly suited to the treatment of gangrenous and severely traumatized tissues, the infection being also combated by the usual antiseptic measures, iodoform, etc. The method is also suited to the after-treatment of skin grafting. The grafts can be covered by a fine wire netting which may be fastened to the surrounding skin by collodion. The part is then placed in the sand bath, the fine netting holding the grafts firmly to the wound surface. The part may be taken out of the bath at night and the netting covered with a dry compress without attempting to wash away the sand which has passed through the netting on to the wound surface. On the following morning the compress can easily be removed.

The Operative Treatment of Varicocele.—NILSON (*Zentralbl. f. Chir.*, 1911, xxxviii, 597), during a service of seven years in a marine hospital, treated 65 seamen suffering from varicocele. All of these, with one exception, were operated on at their own request, on account of pain. In only the one case was the operation performed on account of sexual disturbances, which does not agree with the generally accepted opinion on this point. Nilson did the operation in the inguinal canal because he could better avoid the spermatic artery and, therefore, any resulting interference with the nutrition of the testicle. The constituent structures of the spermatic cord can be easily seen in this situation. It has been shown that if in operations in which a portion of the pampiniform plexus is excised, the stumps are brought together in direct contact, the vessels unite and a secondary reëstablishment of the lumina may occur from the vasa vasorum. Recurrences of the varicocele, however, are frequent. In his cases Nilson did the following operation: An incision was made along the inguinal canal as in the Bassini operation. When the external spermatic vein was varicose, a piece of it was excised. The internal spermatic vein, into which the pampiniform vein empties, was isolated and ligated high near the peritoneum. The lower vein stump was then separated below from its sheath, which is easily done because no branches are given off. It was then drawn upward until the testicle was felt near the symphisis pubis. In order to fix the testicle in this position, the long lower vein stump was passed between the lower bundles of the aponeuroses of the internal oblique and transversalis muscles and tied in a knot around the lower separated bundles. This knot was further secured by suturing the end of the vein stump to Poupart's ligament. The external oblique and the skin and fascial wounds were then closed by suture. The ends of the two vein stumps are so separated that a re-establishment of the blood paths is out of the question and the testicle is securely suspended. The operation can be performed in fifteen minutes and under local anesthesia without pain, the ilioinguinal and external spermatic nerves being injected with cocaine. Thirty-seven cases were later investigated; 2 after six years; 3 after three to four years; 3 after two to three years; 5 after one-half to one year; and 21 after three to six months. Thirty-three were completely cured; 3 were much improved; and only one after seven months was not free of pain. All those which were not investigated were discharged from the hospital, free of symptoms after a two weeks' convalescence.

The Prognosis of Dislocations of the Shoulder and of Posterior Dislocations of the Elbow.—Schmidt (*Deut. Zeitschr. f. Chir.*, 1911, cix, 20) investigated the material treated in the Kiel surgical clinic from 1900 to the end of 1908. Schulz had already reported on similar investigations from Küttner's clinic at Breslau, and had found the results very unsatisfactory. Schmidt found that 92 cases had been treated in the Kiel clinic in the time stated, of which 56 could not be located, and 9 others excluded because of complications such as fractures, paralyses, and recurrent dislocations. There remained 27, in 22 of which the final results were very good. A limitation of abduction of five to ten degrees was disregarded, as it was found to amount to practically nothing in interfering with the usefulness of the arm. There were 6 of these, and the patients were well satisfied with the results. Crepitation in the joint, without limitation of movement or other disturbances, was found in 26 per cent. of the cases. Slight atrophy of the scapular muscles or of the deltoid was found in 29.6 per cent., but the results were otherwise good. Atrophy was present in all the cases with bad results, with one exception. The age of the patients had nothing to do with the prognosis. A good result was obtained in 81.4 per cent. of the cases. In those cases in which the results were poor, the after-treatment was begun after two to three days and consisted in massage, gymnastic exercises, and methodical exercises with medico-mechanical apparatus. The best treatment consisted in keeping the shoulder at rest for ten to fourteen days, by a sling from the wrist and fixation with a loose bandage. Gentle massage was employed to improve the circulation and favor absorption of the extravasated blood. With this treatment the prognosis of an uncomplicated dislocation of the shoulder should be regarded as favorable. With regard to the posterior dislocations of the elbow, the general tendency to give a good prognosis is hardly justified. Forty-six cases had been treated from 1900 to 1908, inclusive. Four of these were excluded because of various complications and 22 more because they could not be located. Of the 20 remaining, 1 was unreduced. Of the 19, in 13, or 69 per cent., the return of function was complete, except for about 10 degrees of limitation of flexion and extension, which was not regarded as interfering with the usefulness of the arm. In 4 cases the disturbances were serious in all the movements of the elbow-joint, in 1 the ankylosis being complete. Schmidt warns here, as in the shoulder, against too early and too energetic therapy, and advises rest of the joint for about the first two weeks.

A Contribution to the Question of Coxalgia, Coxa Vara, and Juvenile Osteo-arthritis Deformans.—Levy (*Deut. Zeitschr. f. Chir.*, 1911, cix, 205) says that a coxalgia can be associated with an existing coxa vara and a picture of a contracted coxa vara may be presented. After an epiphyseal separation in a tuberculous individual with coxa vara, a tuberculous infection can arise secondarily in the hip-joint at the site of the focus of lessened resistance. Before attempting to correct forcibly a coxa vara, the possibly existing tuberculous coxitis must be excluded with certainty. When lung tuberculosis exists in a case of coxa vara, forcible correction should be avoided as much as possible. The affection designated as juvenile osteo-arthritis deformans is not

the same as the usual arthritis deformans. It is due to disturbances in the region of the epiphyseal cartilage, just as in coxa vara. The great clinical and anatomical similarity between these two conditions justify the name of coxa vara capitalis in the place of juvenile osteo-arthritis deformans. A large percentage of the cases of so-called osteal tuberculosis of the hip-joint, which are cured with normal or very good mobility, are really not tuberculous, but cases of coxa vara capitalis.

Injury as a Causative Factor in Cancer.—COLEY (*Annals of Surgery*, 1911, liii, 615), as the result of a careful study of the evidence based upon 1200 personal observations, believes that local trauma, from chronic irritation to a single local contusion, is not infrequently the direct exciting cause of malignant tumors of all types. That a single local injury may cause a carcinoma as well as a sarcoma is no longer open to speculation. The cases that he has submitted fulfil all the conditions necessary to establish a definite casual relationship between a single trauma and the development of a cancer. This relationship depends in no way upon our ability to offer a scientific explanation for it; nor does it depend upon the acceptance of any one of the various hypotheses as to the etiology of cancer. It can be equally explained, whether we accept the extrinsic or intrinsic origin of malignant tumors. The medicolegal aspect of this question is as yet in a most unsettled state. While we must admit that trauma often plays an important causative role in the formation of malignant tumors, this relationship must be clearly and definitely established, according to principles and conditions very similar to, if not quite so exacting as, those laid down by Segond, before any legal liability can be admitted.

Gastro-enterostomy as Shown by the X-rays.—HÄRTEL (*Deut. Zeitschr. f. Chir.*, 1911, cix, 317) says that while the physiology after gastro-enterostomy has been thoroughly studied in animals, the conclusions drawn from such studies cannot be applied with force to ulcerated stomachs in men. Härtel made his studies in patients with particular reference to the late results. Some were studied four months after operation, some as late as three years, the average being one and one-half years. All of them had suffered from benign affections of the stomach. Twenty-two cases were thus studied with the aid of the *x*-rays. The anastomotic opening participated in the evacuation of the stomach in every case, the pylorus in only a part of the cases. The bismuth food introduced into an anastomosed stomach was retained until it filled the stomach entirely. Gradually the evacuation took place, beginning earlier and being earlier completed than in a normal stomach. Aside from that which took place through the pylorus, the evacuation occurred periodically and synchronously with the peristaltic movement of the body of the stomach. Without the development of a new sphincter apparatus, a gastro-enterostomy acts in a manner similar to the physiological action of the pylorus. It does not work as nicely as the pylorus, but has something of the relation to it that a deficient heart opening has to a normal one, and can be seriously crippled if the open pylorus works vigorously. The kind of pathological process affecting the stomach may involve the mechanism of evacuation after a gastro-enterostomy. This evacuation through the gastro-

enterostomy opening is not purely a mechanical process according to the law of gravity; but is influenced by the physiological laws of the stomach movements. The capacity of the stomach for active movement exceeds that necessary for evacuation through the gastro-enterostomy opening, and is capable of increasing the tone of the stomach in ectasia after gastro-enterostomy. The prognosis depends in good part upon the indication for operation. Other factors affect it, especially accessory diseases, by causing later troubles. The mechanism of evacuation is also affected by the indication for the gastro-enterostomy, since the activity of the pylorus is important for the later function. When it is done for a stenosed pylorus, the new opening, while not ideal, for practical purposes fulfils the role of the pylorus. Spastic conditions of the pylorus are relieved by gastro-enterostomy. When it is done for an ulcer of the middle portion of the stomach, it frequently happens that the food is not diverted sufficiently through the new opening and in great part goes through the pylorus as before the operation. Frequently internal treatment will be necessary after the gastro-enterostomy.

THERAPEUTICS

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.,

PROFESSOR OF APPLIED THERAPEUTICS IN THE COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK.

The Treatment of Acidosis.—LICHTWITZ (*Therap. Monatshefte*, 1911, xxv, 81) advocates sodium citrate in place of sodium bicarbonate for the treatment of acidosis. He says that sodium citrate is practically tasteless and may be added to food and also given dissolved in water with the addition of lemon juice. When given this way it makes a very pleasant drink. Sodium citrate causes much less disturbance to the digestion than sodium bicarbonate. He has given sodium citrate up to 50 grams a day, and it has not caused diarrhea, that sometimes results from large doses of sodium bicarbonate. He also adds that it is theoretically possible to give sodium citrate solution by subcutaneous injection, while the strong alkaline reaction of a sodium bicarbonate solution prevents its use subcutaneously. However, he has had no personal experience with subcutaneous injections of sodium citrate.

Silver Nitrate Irrigations in Treatment of Infectious Diarrhea in Children.—SMITH (*Boston Med. and Surg. Jour.*, 1911, clxiv, 298) writes concerning the treatment of infants ill with infectious diarrhea by colon irrigations of 3 per cent. silver nitrate. A cleansing irrigation with sterile water is given before the silver nitrate solution. After the preliminary cleansing has been completed, one pint of a 3 per cent. silver nitrate solution is allowed to run into the colon and the tube

then withdrawn. Some of the solution is retained. Smith says that there was no marked evidence of discomfort following the injections. Thirty-two cases were treated by this method. Three infants showed a rather severe temporary reaction. These infants were very sick, and Smith doubts if the reaction was any greater than it would have been had they received the simple irrigation without the subsequent silver nitrate injection. The treatment was given early in the disease in 22 cases; 11 of these were markedly improved and 7 slightly; 3 out of 6 patients who received the treatment late in the disease were benefited. Smith believes that blood and pus disappear from the stools more rapidly and that the course of the disease is shortened.

The Vaccine Treatment of Typhoid Fever.—WALTERS and EATON (*Med. Record*, 1911, lxxix, 797) report 35 cases of typhoid fever treated by vaccines. This report supplements previous communications, in which they claimed that the vaccine treatment of typhoid fever reduced the mortality, shortened the length of the disease, and rendered relapse less liable to occur. They give a brief synopsis of any unusual features of each case, with the accompanying temperature charts. No aggravation or injury of any kind was demonstrable as a result of any inoculation. They believe that small dosage is an important factor in successful treatment particularly in making the second and subsequent inoculations somewhat smaller than the first. The more serious the clinical condition of the patient the smaller the dose employed. This was apparently shown in one case that was considered to be all but hopeless. Here daily doses of one or two million bacilli were followed by a gradual change for the better in the clinical symptoms.

An Analysis of the Clinical and Serological Results Obtained in the Treatment of Syphilis with Salvarsan.—FORDYCE (*New York Med. Jour.*, 1911, xciii, 861) reports 175 cases of various stages of syphilis treated with salvarsan. He is firmly convinced that this remedy is of great value, although the original claim of a therapia sterilisans magna is no longer entertained by those most familiar with its use. Fordyce believes that the chief question to determine at present is the standardization of its method of administration. It has now been quite conclusively established that a repetition of the dose in practically all stages of the disease is desirable. He recommends the following procedure for the treatment of syphilis: An intravenous injection followed by a course of mercurial inunctions or injections over a period of from four to six weeks. At the expiration of this interval a second intravenous injection of salvarsan and a subsequent course of mercury. At the end of this treatment a rest of a month and a Wassermann reaction. If it is negative, the patient is observed for one or two months and a second serum reaction is made. As long as it continues negative no treatment is indicated. Should it become positive a third intravenous injection of salvarsan supplemented by a course of mercury would be the best procedure to adopt. It seems to Fordyce that this method offers to the patient a far better chance of a rapid and permanent cure than the use of mercury alone. Should the reaction become negative after a single injection of salvarsan, it may be well to act on the advice of Ehrlich and repeat the injection, because of the possibility

of incomplete sterilization with the encapsulation of a few spirochetæ in the bony canals and the danger of recurrence in the nerves of special sense. Fordyce says that the aim of all treatment is to bring about a clean and clear negative Wassermann reaction which continues so for about a year. He believes that the majority of patients are inefficiently treated and that their treatment is not sufficiently controlled serologically. Fordyce thinks that the following conclusions are justified from his experience with salvarsan: In the primary and secondary stages of the disease two doses of salvarsan combined with active mercurial medication materially shortens the duration of the disease. The remedy certainly limits the contagious period of the infection, and in malignant cases its effects are nothing short of miraculous. It produces a rapid healing of the existing lesions, increases the patient's appetite, and improves the body nutrition. It is possible that in such cases we have a different strain of organisms which are more easily influenced by salvarsan than by mercury or potassium iodide. It is rather paradoxical, but it is nevertheless true, that the more serious the case and the more cachectic the individual, the more rapid are the effects of salvarsan. From a study of this disease extending over a period of thirty years Fordyce believes that he has seen more rapid and brilliant results from salvarsan in this class of cases than he has ever seen from the administration of the classical drugs.

The Use of Digipuratum in Heart Disease.—Boos, Newburgh, and Marx (*Arch. Int. Med.*, 1911, vii, 551) report their experiences with the use of digipuratum in cardiac disease. Digipuratum is a purified product derived from digitalis leaves with the elimination of digitonin, that is responsible for the irritant action on the gastro-enteric tract. This purified digitalis extract is a yellow liquid. It is standardized physiologically and is then taken up with sugar of milk to form a powder. The powder obtained is further diluted with sugar of milk until the resulting product has a definite and constant pharmacological strength. For greater convenience of dosage digipuratum is usually dispensed in tablet form, each tablet containing 0.1 gram of the powder corresponding to the average strength of a single dose (0.1 gram) of the crude powdered leaves. These tablets have an agreeable vanilla flavor and are taken readily by all patients. Digipuratum has now been in use at the Massachusetts General Hospital for over a year, and more than 180 cases of primary heart disease or secondary cardiac involvement have been treated with it. The effect on the urinary output has been very prompt in most instances. There was not a single case of vomiting or diarrhea; in fact, the vomiting of a number of cardiac patients at entrance was promptly stopped by digipuratum. Cumulative poisoning has never been observed. In one or two instances sudden drops of forty or more beats in the pulse rate occurred, but no disagreeable consequences followed in any case. It must be borne in mind, however, that digipuratum is a digitalis preparation, and that as such must necessarily have a tendency to produce poisoning by cumulation. In the case of digipuratum this tendency is merely much diminished, so that it is possible by means of this drug to push digitalis therapy in a manner heretofore unknown. This preparation has the advantage of being a more stabile preparation than other digitalis preparations and consequently dosage is more accurate.

A Safe and Rapid Method for Antityphoid Vaccine.—Fletcher (*Jour. Amer. Med. Assoc.*, 1911, lvi, 1016) describes in detail the method of administering antityphoid vaccines in the United States Army. In brief, the method consists in making three subcutaneous injections, at ten-day intervals of a Rill culture of Bacillus typhosus. The vaccine is contained in sealed ampoules, the standard content being 1,000,000,000 dead bacilli per cubic centimeter. The injections are made in the posterolateral aspect of the arm. The initial dose is 0.5 c.c. and the two succeeding doses are 1 c.c. each.

The Injection of Drugs, Especially of Salvarsan, into the Lumbar Muscles.—Meltzer (*Med. Record*, 1911, lxxix, 515), on the basis of experimental observations, suggests that injections of drugs, especially salvarsan, should be made into the sacrospinal muscle. He believes that such injections are practically not inferior to intravenous injections. He says that the sacrospinal muscle presents anatomically an exceptionally well isolated large compact mass, densely packed with fine muscle bundles. Any solution or suspension injected strictly into the mass will remain there and not affect locally the adjacent tissues. Experimentally it was shown that the absorption from the muscle is superior to that from the subcutaneous tissue. Clinically it was established in a limited number of cases of syphilis that salvarsan injected into this muscle excites a fairly rapid, unmistakably beneficial influence upon secondary and tertiary manifestations of syphilis and upon the Wassermann reaction without causing pain and other ill effects deserving any serious consideration. Meltzer gives the technique of these intramuscular injections in detail.

The External Application of Magnesium Sulphate in the Treatment of Erysipelas.—Choksy (*Lancet*, 1911, clxxx, 300) reports 72 cases of erysipelas treated by wet dressings of a saturated solution of magnesium sulphate in water as originally advocated by Lucker. Lucker has described this method of treatment in 19 cases of erysipelas complicated with alcoholism, acute nephritis, myocarditis, pneumonia, etc., with but three deaths, and in 35 uncomplicated cases without a single death. This treatment has been applied by him and others to nearly 700 cases of various forms of inflammation with uniformly good results, and Lucker claims the following advantages for it: (1) Magnesium sulphate is easily obtained, is inexpensive, non-toxic, and is easy of application. (2) The patient promptly obtains relief from the distressing local symptoms. (3) The temperature rapidly falls to normal, usually during the second twenty-four hours, thus eliminating possible complications from a more prolonged fever. (4) There is no necessity for internal medication in uncomplicated cases, the only treatment being a milk diet until the temperature becomes normal. Choksy found in his series of cases, that in almost every instance the immediate effects of the application of magnesium sulphate solution were extremely beneficial. Pain and swelling abated, fever decreased, and extension of the infection was controlled in all but 6 cases. Many of the cases were advanced and neglected, and consequently the results have not been so uniformly good as those reported by Lucker. In 6 cases the inflammatory process extended so rapidly that in spite of this treatment antistrepto-

coccus serum was injected; only 1 of these cases recovered. Excluding the above and 7 moribund cases, there remained 59 cases, with 13 deaths and 46 recoveries. equivalent to about 22 per cent. as compared with about 16 per cent. in Lucker's complicated cases.

The Treatment of Pernicious Anemia with Salvarsan.—Bromwell (*Brit. Med. Jour.*, 1911, 2619, 547) reports 2 cases of pernicious anemia treated with salvarsan. The first case received one injection of 0.2 gram and three injections of 0.3 gram. The hemoglobin rose from 50 per cent. to 78 per cent., and the red blood cells increased from 1,720,000 to 3,580,000. The second case received four injections of 0.3 gram of salvarsan and there was an increase of hemoglobin from 52 per cent. to 80 per cent., with an increase of the red blood cells from 1,760,000 to 4,020,000.

Adalin, a New Hypnotic.—Froehlich (*Berl. klin. Woch.*, 1911, xlviii, 18) has employed adalin, a new sedative and hypnotic in a large number of cases and says that its action is prompt, safe, and lasting. Adalin is a combination of bromine with diethyl-acetyl-urea and may be obtained in powder or tablet form. He used it in doses of 1 gram; if a second dose was necessary, 0.5 gram was given. It is best given with some warm drink. Froehlich noticed no cumulative action from its use, and there seemed to be no tendency for the drug to lose its effect when taken over long periods of time. He gives the details of the cases in which he used it, and found that it induced sleep when other hypnotics had failed.

Serum Treatment of Hemophilia.—Weil (*Rev. Française de Méd. et de Chir.*, 1911, viii, 23) reports several successful cases of the serum treatment of hemophilia. One patient suffered from severe attacks of spontaneous bleeding since infancy, at intervals of not more than three months. Treatment was started during an attack of hematuria, and since that time the patient has had no further attack of spontaneous bleeding. In the first stage of the treatment excessive hemorrhage following cuts did not occur, but recurred whenever the intervals of injection were lengthened. Four years later the patient suffered at intervals from some articular pains, but otherwise was reasonably well. The bood which formerly required four and one-half hours to coagulate now does so in forty to fifty minutes. A member of the same family suffered from the same condition and under treatment showed a similar improvement. Weil also mentions the case of a child, aged seven years, who was very much benefited. The cessation of bleeding in children was followed by a rapid increase in weight. The cases treated by this method are of course as liable to the danger of anaphylaxis as are any others continuously treated by serum therapy. Walters and Eaton used horse serum and antidiphtheritic serum in doses of 20 c.c., given subcutaneously and repeated the injections every two months.

PEDIATRICS

UNDER THE CHARGE OF
LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.,
OF PHILADELPHIA.

Total Congenital Absence of the Teeth.—WIETING (*Deut. med. Woch.*, 1911, xxxvii, 504) reports the total absence of the teeth in a boy, aged twelve years, with slightly retarded physical development. The condition was congenital. The lips and orbicularis oris muscles were hypertrophied and the food was macerated mostly by the tongue. There was entire absence of the alveolar process and the matrix for tooth development. This was verified by the *x*-rays. There was no other abnormality of the body, nor of the hair or nails. But few similar cases are recorded in literature. Partch has reported several cases in which there was an absence of teeth on one side, and a corresponding absence of hair on the same side of the head. In this case Wieting could find no abnormality in any other respect, and there was no evidence of cretinism, syphilis, or rickets to throw any light on the etiology of the case.

Chronic Lung Affections in Children.—SAWYER (*Brit. Jour. Child. Dis.*, 1911, viii, 151) describes the chronic lung affections in children under two heads—bronchial catarrh and a mild form of bronchiectasis. These chronic conditions are difficult to diagnosticate owing to an absence of abnormal physical signs. In cases of chronic bronchial catarrh the children may have a cough for many years, with scarcely any abnormal physical signs. They are usually delicate and anemic, but show no signs of cyanosis. There is no clubbing of the fingers or dyspnea, but there is a tendency to have many colds. The cough has existed from infancy in many cases, or from some former febrile attack. Expectoration is usually absent. A few rhonchi are heard at times when the patient has a cold, but no coarse lesions are demonstrable. In establishing a diagnosis the most important part of the history is the duration of a cough for several years; this rules out tuberculosis. Recovery often occurs with the growth of the child. Enlarged tonsils, adenoids, and neuroses must be excluded in making the diagnosis. Chronic bronchitis in infants is rare. It is, however, constant in scoliosis. In chronic bronchitis the patients resemble the first group alluded to, but usually have cyanosis, more frequent cough occurring in paroxysms, and usually some phlegm. Rhonchi and coarse rales are present in the lungs, which show signs of emphysema. The physical signs in the lungs are bilateral, and there is no wasting or fever. The treatment is similar to that prescribed in adults, and most benefit is derived from potassium iodide. Bronchiectasis in a mild form is found quite often, but severe types are rare. As the child grows older the bronchi often resume their normal size. Here there is a constant cough and a large quantity of phlegm with a slightly fetid smell. There

is usually slight clubbing of the fingers, but not much wasting or nocturnal fever. A few rhonchi are usually heard at the bases. There is no consolidation, as a rule, and the breath sounds and percussion note are normal. The rare, severe forms show moderate wasting, fever, cyanosis, a large amount of phlegm, and definite clubbing of the fingers. There may be contraction of one side of the chest with displacement of the heart toward that side and emphysema of the opposite lung. There may be septic cachexia. The treatment, besides general hygiene, consists of continual inhalation of drugs on a "Yeo" inhaler. Four drops of a mixture of creosote, ℥ij; carbolic acid, ℥ij; tinct. iodine, ℥j; spiritus etheris, ℥ij; and spiritus chloroformi, ℥ij, are placed on the sponge every two hours during the day, and three times at night. Phthisis of the adult type is very rare in children under fifteen years.

Ephemeral, Traumatic Glycosuria in the Newborn.—Erich Hoeniger (*Deut. med. Woch.*, 1911, xxxvii, 500) reports 4 cases of transient glycosuria in newborn infants delivered with forceps. In all the cases the family history of the mother showed no diabetes. Sugar was present in the urine of the four infants up to the second, third, and fourth day after birth, when it disappeared. The deduction follows that the sudden trauma of the forceps on the child's head is the cause of the glycosuria, since normally born children, even where the labor is very difficult and the head subject to much pressure, do not show any signs of glycosuria. These cases are reported to encourage investigation along this line, especially to determine whether these temporary glycosurias may be the beginning of a diabetes mellitus later on in childhood.

Petechial Hemorrhages from Artificial Stasis as a Diagnostic Help in Scarlet Fever.—Leede (*Münch. med. Woch.*, 1911, lviii, 293) describes a diagnostic point in scarlet fever. During an epidemic, while taking blood from veins in the arm of scarlet fever patients, he noticed that soon after compressing the arm with a bandage hemorrhages appeared in the skin distally from the bandage. These petechiæ always appeared in those suffering from scarlet fever, but did not seem to occur in those suffering from other diseases. A study was made of this phenomenon, using a rubber tourniquet on the upper arm sufficiently tight to cause venous stasis, but not compressing the arterial flow. Almost without exception in several hundred cases of scarlet fever tested, more or less extensive hemorrhage appeared under the skin. Control tests in conditions other than scarlet fever showed occasional slight tendency to similar petechiæ, although this was far from constant. Even in healthy individuals this reaction was noted in a very few cases. It was determined that the grade of the reaction depended on the resistance of the capillaries, which in scarlet fever is much impaired by the toxins of the disease. To determine this grade of reaction a modified Riva Rocci blood pressure apparatus was used. The systolic and diastolic pressures were determined. The cuff was then filled with air until a pressure was reached just below the diastolic pressure, usually 45 to 60 mm. of mercury. This pressure was maintained for from five to twenty minutes, and the skin watched for petechiæ, which develop in from five to twenty minutes, usually at the

bend of the elbow. In this way the veins are blocked and the arteries are not compressed, and the stasis is developed from *vis a tergo*. In other conditions it was exceptional to get a result similar to this; only in measles were results noted which at all approached those found in scarlet fever. This phenomenon appears fairly early in the disease, at least with, and occasionally before, the appearance of the eruption. The capillary susceptibility diminishes as convalescence goes on, and while the majority showed the phenomenon up to the forty-second day, in a large number of cases the reaction could not be elicited after the twenty-first day of the disease. Females seem to lose the reaction earlier than males. Only in one clinically typical case of scarlet fever did this reaction fail, and this was probably due to subcutaneous fat. To make this test place a rubber bandage around the upper arm, causing venous distention, but not occluding the arterial pulse. In from five to fifteen minutes the bandage is loosened. The tender skin at the angle of the elbow is then put on the stretch to determine a sprinkling of dark petechial hemorrhages.

Confirmation of Rumpel and Leede's Phenomenon in Scarlet Fever.—BENNECKE (*Münch. med. Woch.*, 1911, lviii, 740) confirms the findings of Rumpel and Leede in regard to petechial hemorrhages from artificial stasis in scarlet fever. He has noticed this phenomenon frequently in making blood examinations in scarlet fever, and found it positive in 26 out of a series of 32 cases. In doubtful cases in children, where the throat signs were suspicious, a positive result was always confirmed by the development of typical scarlet fever. He found the result positive in 1 case of measles and in 2 cases of high arterial tension (230 mm.), in which latter cases, however, the spots spread over the forearm and hand and remained visible from two to three weeks. The typical lesions were found only about the elbow, the small, pinhead-sized hemorrhages disappeared quickly, the larger sized petechiæ disappearing in from one to two days. In the two cases of high arterial tension the petechiæ became pigmented. The negative result of this test speaks against scarlet fever. The positive result does not absolutely prove scarlet fever.

Edema during Gastro-intestinal Conditions in Infants.—HUGH ASHBY (*Practitioner*, 1911, lxxxvi, 686) calls attention to the edema of the feet and hands which often develops during a gastro-intestinal disease in infants. The edema usually appears toward the end of an attack of gastro-intestinal catarrh, when the vomiting and diarrhea are getting better. It does not develop during the acute stage. The edema is bilateral and attacks the dorsum of the hands and feet, and occasionally the face becomes puffy, giving the appearance of a renal condition. The heart and lungs are usually found normal. The urine is high-colored, acid, and foul-smelling, and contains a few leukocytes, but no blood or casts. Small amounts of albumin are found at times, but while there is no nephritis, there is renal inadequacy and the child is suffering from toxemia. Necropsy shows no change in the kidneys, except slight cloudy swelling in the tubules. The blood in these cases shows a high proportion of hemoglobin and a slight increase in the number of red blood cells. The circulation is not especially feeble.

Edema in the course of gastro-intestinal diseases is a serious symptom, although quite a large number of cases recover. The chlorides are not diminished in the urine, as they are in cardiac diseases with edema. In the treatment of this condition, besides general hygienic measures, it is important in feeding to exhibit a high percentage of proteid and a low percentage of fat and carbohydrate. Potassium citrate is the best diuretic. Tincture of digitalis in $\frac{1}{10}$ drop doses every two hours for a short time is often of service.

OBSTETRICS

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.,

PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

Our Recent Advances in Obstetric Surgery of Proved Value.—Those who enjoy a critical review of modern methods will find much of interest in a paper by ROTH, read before the Gynecological Society of Dresden (*Zentralbl. f. Gynäk.*, 1911, No. 7). He reviews the literature recently published, advocating the early getting up of puerperal patients. Krönig has enthusiastically advised the early rising of puerperal patients, even in cases which ordinarily would seem to demand especial caution. For this practice he claims a reduction of mortality to 4.3 per cent., and in 1000 patients no case of thrombosis. The other side of the picture is presented by Frommé, who allowed a primipara, aged nineteen years, to get up for one hour on the second day of the puerperal period. This was followed by fever lasting for four days, and the development of embolism, which ended the patient's life on the eleventh day. Roth believes that we should not abandon the custom of keeping puerperal patients in bed from seven to eight days. He finds no adequate reason for changing this practice. The treatment of placenta prævia by section is also reviewed, and the comparative mortality of section and other methods of treatment is stated. By non-surgical methods, a maternal mortality of 6.6 per cent. is reported, and a fetal mortality of 30 per cent. Roth would agree with Veit in declining to adopt Cesarean section for placenta prævia on the ground that no better results will be obtained than follow other methods of treatment. The suprasymphyseal section receives also an adverse verdict, and the statement is made that in two years after its enthusiastic introduction it has made no essential headway. The field for vaginal section is thought to be an exceedingly limited one. It is always beneficial to consider critically any method of operation which is an essential change from methods which have been proved to be of value. In the treatment of placenta prævia by section, the critic does not distinguish between central placenta prævia and other varieties. In partial, marginal, and lateral placenta prævia the rupture of the membranes and the stimulation

of uterine contraction is sufficient in many cases. In others, the introduction of a bag through the ruptured membranes, and through a portion of the placental substance, has been proved to be of value, but in complete placenta prævia, with mother and child in good condition, the classic Cesarean section, when promptly done in the hospital, gives results superior to other methods of treatment.

Spontaneous Gangrene in both Ischiorectal Fossæ.—LEYZEROWICZ (*Zentralbl. f. Gynäk.*, 1911, No. 8) reports the case of a woman, aged twenty-two years, who gave birth at full term to a living child in spontaneous labor, with normal expulsion of the placenta and without laceration. On the seventh day of the puerperal period the patient's temperature rose to 104°. She had no abdominal pain, but complained of severe pain in the region of the sacrum. The lochial discharge was normal, and the bowel movement normal. The uterus was twice curetted without result. Later, redness developed in the region of the anus, and preparations were made to practise incision and drainage. The swelling opened spontaneously, and purulent fluid escaped. The opening was dilated, and necrotic material and decomposed fluid escaped. A considerable portion of tissue had become gangrenous. A second opening in the other ischiorectal fossa developed, which was similarly treated. The patient made a tedious recovery.

The Repair of Vesical Vaginal Fistula after Hebostiotomy.—CHRISTOFOLETTI (*Zentralbl. f. Gynäk.*, 1911, No. 8) reports the case of a patient who had a very small fistula communicating with the neck of the bladder near the urethra. Two operations were performed to close this fistula, but unsuccessfully, and pregnancy supervened, which was terminated by Cesarean section. Eight weeks after operation the fistula was closed by separating the bladder from the pubic bone and bringing together the edges of the fistula with two rows of catgut stitches. From this time the patient made a good recovery. He also reports the case of a patient who four years previously had hebostiotomy performed, the puerperal period being complicated by fever. There was no laceration of the vagina, but the patient did not retain the urine. Operation was performed, as in the first case, by loosening the bladder from the pubic region and bringing the edges together in layers. A good result followed.

Myoma Complicating Pregnancy.—SCHAUTA (*Zentralbl. f. Gynäk.*, 1911, No. 8) reports the case of a patient, six months pregnant, who suffered from pain in the left side of the abdomen. This became so severe that the patient willingly consented to operation. A tumor as large as a kidney was found upon the left side of the pregnant uterus. Incision was made along the border of the left rectus muscle and a myomatous tumor found attached to the uterus by a broad pedicle. The tumor was removed and the tissues brought together. Pregnancy proceeded without interruption. On examining the tumor it was found to be a necrotic myoma. The necrotic process evidently had something to do with the patient's pain. In discussion, Fabricius, in 578 operations for myoma, had 9 cases complicated with pregnancy, 1 of these having an ovarian cyst in addition. In 223 cases, 5 opera-

tions upon myomatous tumors were performed because of pregnancy as a complication. These cases were treated by supravaginal amputation of the uterus. He also reports the interesting case of a patient, aged thirty years, who at the fourth month of pregnancy had the uterus increase so rapidly that at six months it filled the entire abdomen. Abortion occurred, with the expulsion of the fetus, and upon examination a soft tumor as large as the head of a child was found in the right wall of the uterus. The placenta had been partially attached to this tumor. The tumor steadily decreased in size, and eight days after the abortion could scarcely be detected. A subsequent pregnancy occurred which proceeded normally, with normal labor. At this time scarcely a trace of the tumor could be found. A second case is also reported in which the tumor grew very much less after the birth of the child. Peham, in 117 cases, had 8 complicated by pregnancy. Two were treated by conservative methods, removing the tumor without interrupting the pregnancy. In one case the tumor was so large that operation was necessary.

Advanced Extra-uterine Pregnancy.—WAGNER (*Zentralbl. f. Gynäk.*, 1911, No. 8) reports the case of a patient who came to his clinic in a pregnant condition complaining of almost constant pain in the abdomen. When pregnant about six months the fetal movements ceased, and the abdominal pain grew very much less. On examination, the abdominal tumor extended two fingers beneath the tip of the sternum. The head of a full term fetus could be palpated in the abdomen, and the pelvic tissues showed signs of pregnancy. The placenta could be made out as a comparatively soft mass attached to the uterus by a pedicle. Operation was performed under lumbar anesthesia and the tumor was covered by the omentum which was adherent to the pelvic brim. After this was loosened the fetus was found in a grayish-white capsule. The tumor, which was supposed to be the placenta, was a hydrosalpinx as large as an orange. On the right side the tube terminated in a grayish-white hard tumor as large as two fists, which was attached to the sac containing the fetus. This tumor was adherent to the surrounding tissues. The appendix was adherent to a smaller tumor. The ovaries were normal, and the uterus and left ovary were left. The hydrosalpinx, fetus, and placenta were removed. On examination, it was found that the fetus had now become a lithopedion. The placenta showed calcareous changes, and was contained in a sac composed of the tube. The fetal sac was connective tissue developed in a plastic peritonitis, and the sac was so adherent to the fetus that some of the hair from the scalp was firmly fixed in the sac. There was no amniotic liquid. The left foot of the fetus was much bent, but otherwise the skeleton was normal. There had been no symptoms of intraperitoneal hemorrhage or acute peritonitis.

Momburg's Method for Controlling the Circulation in the Pelvis.—MOMBURG (*Archiv f. klin. Chir.*, 1911, Band lxxxix, No. 4) again draws attention to the value of his method in preventing pelvic hemorrhage. In 17 obstetric cases he was able to render the uterus practically anemic and bring about vigorous uterine contraction. He

uses from two to four bandages, and, in addition to that passed about the abdomen, he bandages the lower extremities also. Anesthesia may be used, and it is found that less ether than ordinarily is required. The patients recover speedily after anesthesia. The heart action is sometimes weaker than normal and requires stimulation, while in other cases its function is undisturbed. In cases coming to section no injury to the abdominal viscera has been found. The bandages can remain, if necessary, for two hours. If anesthesia is not employed morphine should be given.

GYNECOLOGY

UNDER THE CHARGE OF

JOHN G. CLARK, M.D.,

PROFESSOR OF GYNECOLOGY IN THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

Sacro-iliac Relaxation.—MEISENBACH (*Surg., Gyn., and Obstet.*, 1911, xii, 411) draws attention to the fact that the sacro-iliac joint is, contrary to the older teaching, a true joint, possessing a hyaline articular cartilage, a synovial membrane, capsule, and ligaments, and that it normally permits of a slight degree of motion, varying in individual cases. When the pelvic ligaments have a normal tonus the joint can withstand considerable strain, but when they become relaxed, the joint yields easily, and may cause many marked symptoms ordinarily referred to intrapelvic conditions, but which are not relieved by the gynecological operation which appears to be indicated. Among several causes, however, which may produce a relaxation of the sacro-iliac ligaments, Meisenbach considers pelvic congestion as one of the most important; the condition also often arises after childbirth, especially after a high forceps delivery. Many of these cases receive no relief from gynecological treatment, but if properly diagnosticated and treated they can almost always be rescued from chronic invalidism. Meisenbach considers stereoscopic *x*-ray pictures of considerable value in diagnosticating a sacro-iliac relaxation, whereas in the ordinary plate it usually passes unnoticed. The treatment is a properly applied plaster or celluloid jacket, steel braces, elastic webbing, etc., according to individual indications. Strapping by means of adhesive strips applied firmly from one crest to the other may afford temporary, but only temporary, relief.

Treatment of Cystocele.—BRENNER (*Monats. f. Geburts. u. Gyn.*, 1911, xxxiii, 464) asserts that the only really fixed portion of the bladder is that directly over the anterior cervical wall, and that this is the first part of the bladder to enter into the formation of a cystocele, which, therefore, can only occur when the union between the bladder and the anterior cervical wall has given way. Acting on this anatomical

principle, he suggests the following operation for the treatment of the condition. The bladder wall is exposed through the vagina, and its most prominent portion folded in by two purse-string sutures after Gersuny. A posterior colporrhaphy and perineorrhaphy are performed in the usual manner. The abdomen is then opened by a transverse fascial incision, the peritoneum of the anterior surface of the uterus incised from one ligamentum teres to the other and dissected downward till the turned-in bladder diverticulum and the vaginal wall come into view. The infra-ureteral bladder tissue is now puckered together with one or two sutures, after having previously dissected out bluntly the vesical ends of the ureters, great care being taken in introducing these sutures in no wise to disturb the course of the ureter. In this way a firm point of resistance is secured below the ureters and the midsection of the bladder and above the vaginal vault. Then by means of a transversely placed suture the posterior wall of the bladder is fastened to the anterior wall of the cervix; a second similar stitch is placed somewhat higher up nearer to the fundus of the bladder. The peritoneum is then closed, the uterus suspended to the anterior abdominal wall if necessary, and the wound closed. The author maintains that the result obtained by this infra-ureteral suture cannot be attained from below, as the ureters cannot be dissected out without injuring their attachments, and without dissecting them out there is too much danger of including them in the suture, or, at least, of kinking or injuring them. He considers that almost all of these cases have a retroverted uterus, which of itself calls for the performance of a laparotomy.

Relation of the Thyroid to the Ovaries.—Goodall and Conn (*Surg., Gyn., and Obstet.*, 1911, xii, 457) report an interesting case of a markedly enlarged thyroid in a woman, aged sixty years, associated with chronic pelvic tuberculosis, in which the thyroid swelling completely disappeared after removal of the diseased pelvic organs, and then proceed to discuss in detail the relations between the thyroid and the female genital organs, considering this subject from the point of view (1) of the influence of the thyroid upon the genital system, and (2) of the influence of sexual function upon thyroid activity. Under the first heading a group of cases is quoted from Goodall and Conn's experience in which myxedema or other symptoms of a diminished thyroid were associated with diminished or absent menstrual function. In some of these cases most distressing menstrual molimina, going as far as epileptic attacks, occurred at the time when menstruation should have taken place. In all of these cases a marked improvement followed treatment with thyroid extract, in some instances combined with calcium lactate. To illustrate the influence of hyperthyroidism on the sexual function several caess are quoted from Pinard, in which marked menstrual disturbances, usually of the amenorrheic type, were associated with exophthalmic goitre. In discussing the influence of the sexual function upon thyroid activity, Goodall and Conn call attention to the relief of the symptoms often following a natural or artificial menopause, supposed to be due to a loss of balance between the ductless glands produced by small doses of thryoid extract. An overproduction of the ovarian secretion, on the other hand, seems to

cause hyperactivity of the thyroid gland, so that these two glands are to be considered as neutralizers, not as compensators, of each other. This idea is further borne out by the fact that an enlargement of the thyroid gland takes place at puberty, and frequently at the onset of pregnancy, at both of which times a physiological increase in the internal secretion of the ovary occurs. Goodall and Conn emphasize the fact that the action of any two of the ductless glands should not be considered alone, as all these glands are most intimately associated in function. They consider that the facts already brought out—also that exophthalmic goitre almost invariably makes itself manifest during the years of greatest sexual activity, whereas myxedema and cretinism generally occur before or after sexual life—lend weight to the supposition that the thyroid and ovarian glands are under the control of the same governing centre, or that they depress and stimulate each other into activity.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES

UNDER THE CHARGE OF

J. SOLIS-COHEN, M.D.,

OF PHILADELPHIA

Vincent's Angina Involving the Larynx Exclusively.—ARROWSMITH (*Annals of Otology*, September, 1910) reports this case in a stableman, aged twenty-six and one-half years, admitted to hospital with œdematous epiglottis, arytenoids, and ventricular bands, the dyspnœa which became so urgent as to compel hurried tracheotomy twenty-four hours later. The tube was removed after four days and the suppurating tracheotomy wound was completely healed by the third week. Resuppuration ensued, with a recurrence of hoarseness, dyspnœa, and swelling of the soft tissues of the neck, so that a second tracheotomy became necessary. In less than a month this had to be followed by a thyrotomy and the insertion of a laryngostomy tube for the purpose of keeping the larynx open. At the time of the report the patient was unable to breathe at all adequately upon closure of the tracheal orifice. Arrowsmith also reports a more recent case in a male, aged forty-one years, the exudate from whose throat showed numerous fusiform bacilli and spirilla of Vincent implanted upon a tuberculous larynx.

Diverticulum of the Trachea.—KAHLER (*Annales des maladies de l'oreille, du larynx, du nez et du pharynx*, August, 1910) has reported a case of diverticulum of the trachea, discovered accidentally, in a man, aged thirty-four years, who was being treated for spasm of the esophagus at Chiari's clinic.

Intralaryngeal Actinomycosis.—ARROWSMITH (*Laryngoscope*, October, 1910) describes a case of apparently primary intralaryngeal actinomycosis in a male subject, aged nineteen years. Specimens removed from the mass after careful examination allowed no other diagnosis than that of actinomycosis, but a month later no evidence of actinomycosis could be found in the sputum, while tubercle bacilli, staphylococci, and streptococci were present. The case progressed as one of tuberculosis of the larynx.

Laryngostomy in Perichondritis of the Larynx.—YVANOV (*Annales des maladies de l'oreille, du larynx, du nez et du pharynx*, August, 1910) reports that he has treated the losses of substance of the anterior wall of the trachea in eight instances by the procedure of Mangoldt; but in one case he used successfully the cartilage of the nasal septum in place of costal cartilage. In four of these patients respiration by the normal passages had been completely reëstablished, but the others were still under treatment.

Paralysis of the Recurrent Laryngeal Nerve.—MEAD (*Jour. Amer. Med. Assoc.*, December 24, 1910) reports a case of persistent patency of the ductus arteriosus in which there was fixation of the left vocal band in the median line from pressure on the abductor fibers of the recurrent laryngeal nerve. In a discussion upon this case, Tileston mentioned that von Schroetter, Jr., of Vienna, reported a similar case a few years ago, at that time the only one to be found in literature.

New Oral Speculum for Direct Examination of the Mouths of the Eustachian Tubes and Parts Adjacent.—YANKAUER (*Laryngoscope*, March, 1911) describes and depicts an ingeniously devised speculum which exposes the pharyngeal extremities of the Eustachian tubes and the fossa of Rosenmueller, by direct inspection. With its aid adenoid masses can be seen, and be removed with the straight forceps. The interior of the fossa can be seen, probed, irrigated, and packed. Adhesions can be cut with straight scissors and applications made with an ordinary applicator. A small tube about one-fourth inch in diameter can be introduced into the interior of the Eustachian tube, and its lumen examined down to the isthmus.

Auto-inspection of the Vocal Apparatus during Phonation.—WRIGHT (*Laryngoscope*, March, 1911) describes and depicts a modification of Hays' pharyngoscope with an angular telescopic extension reaching to the eye, by which a person may see with wonderful clearness the operation of his own vocal apparatus while producing sound.

Grave Hemorrhage after Tonsillectomy.—MARSCHIK (*Revue Hebd. de lar., d'otol., et de rhinol.*, April 15, 1911) reports a case in which it became necessary to suture the pillars of one side upon a tampon of odoform gauze, and to tie the external carotid on the other side and follow that by an infusion of adrenalinized physiological serum. The patient recovered. The hemorrhage was attributed to the facts that the patient had nephritis, was in the second day of her menstruation, and had been very indocile under the operation.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

WARFIELD T. LONGCOPE, M.D.,

DIRECTOR OF THE AYER CLINICAL LABORATORY, PENNSYLVANIA HOSPITAL, PHILADELPHIA.

Experimental Contribution to the Study of Cardiac Hypertrophy.—HUGH A. STEWART (*Jour. Exp. Med.*, 1911, xiii, 187) has studied the hypertrophy of the heart of dogs following upon the production of an aortic insufficiency. The aortic insufficiency was made by means of the valvulotone described by McCallum. Following the lesion at intervals varying from five to one hundred and twenty days, the animals were killed and their hearts weighed by the Müller method. Not only was the proportional weight of the whole heart estimated, but that of the auricles, ventricles, and the septum separately. The experiments showed that hypertrophy starts very shortly after the production of the aortic insufficiency, and is well marked at the end of a week, and that all chambers of the heart shared in the increase of weight of the heart. The greatest absolute increase, however, is in the left ventricle. The greatest relative increase is also in the left ventricle, but the auricles show a relative hypertrophy greater than that of the septum or of the right ventricle. This hypertrophy of the right auricle with experimental aortic insufficiency has been noted by others, notably Rosenbach and Senator. The simplest explanation is that the hypertrophy depends upon a relative mitral insufficiency which is so commonly associated in man with aortic insufficiency. The presence of a relative mitral leak could, however, not be established in these experiments, and the explanation seems to depend upon other factors. There is no altered venous pressure in the auricle, but there is a marked increase in the force of the auricular systole, which probably explains the co-hypertrophy of the auricles. During the first week after the production of the aortic insufficiency there is only a very small increase in water content of the heart, and consequently it is not possible that the rapid increase in weight during this time could be due entirely to increased water content of the muscle fibers.

The Passage of Antitoxic Sera into the Cerebrospinal Fluid.—It has been known for some time that crystalloids when injected subcutaneously pass with great difficulty or not at all into the cerebrospinal fluid, and it is probable that the same is true of colloids. LEMAIRE and DEBRE (*Jour. de Physiol. et de Path. Gén.*, 1911, xiii, 233) have made some experiments to test the accuracy of this last statement and to determine, as well, whether the protective property of immune sera injected subcutaneously is demonstrable in the spinal fluid. Dogs were injected subcutaneously with tetanus antitoxin from the horse and the cerebrospinal fluid examined later by the precipitin reaction to see whether it contained horse serum, and by injections into animals to determine whether it had acquired the property of protecting these

animals against tetanus toxin. It was found that only infinitesimal amounts of horse serum passed into the spinal fluid, and that the fluid had acquired very slight protective power against tetanus toxin. The blood serum of the dogs, however, contained large quantities of horse serum albumin and developed marked antitoxic qualities. Experiments were then instituted to see if by any means the foreign serum could be made to pass more readily into the spinal fluid. Morphine was administered to the dogs in doses of one centigram per kilo. Under these circumstances, though the amount of horse serum was not perceptibly increased in the spinal fluid, the antitoxic power was very distinctly increased. At the same time the antitoxic power of the blood serum was reduced. They believe that this is to be accounted for by the dilating action of morphine upon the peripheral vessels. They believe that this action of morphine in lowering the antitoxic effect of the blood serum after subcutaneous injection should be borne in mind when sera are administered to man for therapeutic purposes.

The Value of the Treatment of Acute Articular Rheumatism with the Antipyretics.—MENZER (*Zeit. f. Hyg. und Infektionsk.*, 1911, lxviii, 296) brings up the oft discussed question as to the value of the salicylates in the treatment of rheumatic fever. He believes that the disease is a streptococcus infection originating in the tonsils and upper air passages and thence becoming generalized. The inflammatory processes which arise during the course of the disease, in the joints, heart, and serous membranes are manifestations of an attempt upon the part of the body to combat the local infection in these situations and therefore represent the first efforts at healing. Such local reactions cannot be prevented by any method of treatment, and the question at issue is by what method can complete recovery best be brought about. He uses large statistics gathered from the army to show that treatment by the salicylates does not in the long run further the healing process, inasmuch as the cases treated with antipyretics showed a larger percentage of serious injuries to the heart and a greater number of recurrences. He suggests that the salicylates should be replaced by local and general methods such as are usually in vogue for the alleviation of general and localized infections. Such procedures as warm applications, baths, and general attention to the nutrition of the patient might be supplemented by injection of small doses of antistreptococcus serum.

Notice to Contributors.—All communications intended for insertion in the Original Department of this JOURNAL are received only *with the distinct understanding that they are contributed exclusively to this* JOURNAL.

Contributions from abroad written in a foreign language, if on examination they are found desirable for this JOURNAL, will be translated at its expense.

A limited number of reprints in pamphlet form, if desired, will be furnished to authors, *provided the request for them be written on the manuscript.*

All communications should be addressed to—

DR. GEORGE MORRIS PIERSOL, 1927 Chestnut St., Phila., Pa., U. S. A.

VOL

A
T

SO-C

AMY

THE

PAT

INTR

MAC

DYS

THE

THE

COPYR